### Philippine Obstetrical & Gynecological Society, Inc.

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Vulvovaginitis

Author: Rosendo R. Roque, M.D., FPOGS, FPCS

I. Introduction

The infectious pathology of the vulva and vagina is a commonly encountered problem. Although often considered harmless, it can be the cause of dyspareunia, pain, and even sterility. A correct diagnosis can only be made following a rigorous clinical examination accompanied by a microscopic analysis of the vaginal discharge and its quasi-systematic culturing.

Vaginitis is often associated with vulvar or cervical lesions, which may be of bacterial, parasitic, viral, trophic, or traumatic origin. The etiologic circumstances vary according to the reproductive maturity and genital activity as in puberty, period of reproduction, pregnancy, and menopause.

The vaginal physiology is greatly influenced by the hormones. Thus, in this monograph, the essential notions of the vaginal physiology must be reviewed. The study of clinical aspects of various types of vaginitis allows physio-pathologic deductions to be made and the diagnostic techniques discussed except the treatment, which is much too changeable.

II. Physiopathology

A. Origin of the Vaginal Fluid

There is no gland in the vagina; the fluid, which it contains, results from the transudation of the vaginal wall and is not a secretion. The whitish coating of the walls has a creamy consistency. Microscopically, it is composed of desquamated vaginal cells, polynucleates, and a special bacterial flora.

The cervix and the uterus also contribute to the formation of this fluid. In fact, the interior of the cervix possesses a cylindrical glandular epithelium, which secretes mucus, the abundance of which varies according to the cycle. The maximum secretion is reached at ovulation. The glands of the uterine body also produce seromucoid secretions, which are rarely found in physiologic conditions.

In addition, the vaginal fluid, which flows out of the vestibule contains the non-glandular, serous product of transudation of the vestibule, and the secretions of Skene and Bartholin's glands.

The multicentric origin of the vaginal fluid explains why the vaginal infection is often isolated, rarely combined with a cervical pathology, and the origin of its relapse is often found in Skene's and Bartholin's glands.

B. Properties and Composition of the Vaginal Fluid

The vaginal fluid has a high acidity of about pH 3.8-4.2 due to a high level of lactic acid (2 to 3%). This level varies during a lifetime and is influenced by the presence of glycogen and Doderlein's bacilli.

The vaginal cells liberate the glycogen during their desquamation. As the desquamation is influenced by fluctuation of steroid hormones, the vaginal concentration of glycogen undergoes similar variations: Considerable at birth, maximal during ovulation and almost non-existent during childhood and after menopause. The normal microbial flora also varies with age. From the second week of life to puberty, the vagina contains innocuous flora (staphylococci, streptococci, coli, diphtheroids). At puberty, the lactic bacillus reappears as a result of estrogenic secretion and production of lactic acid, which keeps the vaginal pH at about 3.8-4.2. Hormones also influence the acidity level.

At birth, the pH is the same as the amniotic fluid. As soon as the maternal estrogens are eliminated, the pH rises to 6.0-7.5, a value that remains until puberty and drops to about 3.4-4.2 at the beginning of ovarian activity. From this time on, it varies according to the ovarian cycle.

During ovulation, the pH varies between 3.8 and 4.2; during the premenstrual phase, it rises to 5.5 to reach 6.5 to 7.5; during menstruation, it drops again to its previous values. At menopause, the pH rises to 6.5 to progressively reach alkalinity when the vaginal mucus shrinks. The acidity is microbicidal and helps to keep the vaginal environment normal.

C. Natural Defense Mechanisms of the Lower Genital tract

While a woman's genital tract is in direct contact with the exterior environment and is in close proximity to the urethra and anus, it has a natural defense system:

At the vulvar level:
- An obstacle to penetration by coaptation of the labia.
- A naturally high resistance to infection of the vulva and perineum.
- The secretion of undecylenic acid, fungicidal substance produced by the apocrine glands.

At the level of the vagina:
- The coaptation of the vaginal walls.
- The stratified epithelium devoid of glandular orifices.
- The vaginal acidity.
- The vaginal flora.
At the level of the cervix:
- The bacteriologic power of the mucus.

D. Alteration of the Defense Mechanisms

The efficacy of these defense mechanisms can be changed by:

<table>
<thead>
<tr>
<th>Age</th>
<th>During childhood and after menopause, the vaginal epithelium is thin and fragile. The markedly decreased glycogen level and normal vaginal flora reduce the acidity (pH near 7).</th>
</tr>
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<tr>
<td>Menstruation</td>
<td>Genital infections are encouraged by the alkalinity of the menstrual discharge and the disappearance of cervical mucus.</td>
</tr>
<tr>
<td>Puerperium</td>
<td>Infection is encouraged by the decrease of the organism which is taxed by pregnancy, the relaxation of tissues unequally contaminated by the delivery or abortion, and the reduction of the vaginal acidity by lochia.</td>
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</table>

Other than these, the clinician must conduct a clinical examination of the patient and eliminate:

- Hydrorrhoea and purulent discharge of corporal origin secondary to cervical stenosis or an intra-uterine tumor;
- Leucorrhoea due to an intra-epithelial neoplastic lesion or invasion of the cervix;
- Physiologic leucorrhoea:
  - acellular preovulatory period, having the classic aspect of egg white and starching underwear.
  - Lactescence in premenstrual period, a simple exaggeration of the epithelial desquamation of the vagina.

III. Vulvovaginal Candidiasis

Candidiasis has become one of the most frequent infections provoking leucorrhoea. The specific agent of this infection belongs to the group of Candida. About 65% of vulvovaginitis is due to Candida albicans, +25% to Candida tropicalis and more rarely to Candida pseudotropicalis, Candida krusei and Candida stellatoide. The acute forms are usually due to Candida albicans, while Candida tropicalis is most often observed in chronic forms.

A. Clinical Description

Vulvovaginal mycoses are observed in three aspects: Acute, chronic and saprophytic. Vulvar pruritus is the most precocious and intense symptom. A nocturnal recrudescence is often observed. Certain patients complain of a vulvovaginal burning sensation. The leucorrhoea is thick, granular, with the aspect of curdled milk, sticking to the subjacent mucosa; the mucosa has white spots followed by an edema and a deep red erythema of the labia minora.

These symptoms are seen in only 20% of the patients. In other patients, the erythema spreads to the labia majora, the perineum and the perianal region. Occasionally, it spreads to the subpubic region and the inner side of the thighs. Small vesicles and pustules combined with scratching lesions are also observed when the cutaneous infection is primitive. The vagino-vulval irritation can be at the origin of dysuria, pollakiuria, dyspareunia.

Classification of Vulvovaginal Candidiasis (VVC)

| Uncomplicated VVC | Sporadic or infrequent VVC AND Mild to moderate VVC AND Likely to be Candida albicans AND Non immunocompromised women |
| Complicated VVC   | Recurrent VVC OR Severe VVC OR Nonalbicans Candidiasis OR Nonalbicans Candidiasis |

Updated!

Primary cutaneous candidiasis of vulva. Classical example. Primary skin infections of the vulva are unrelated to the usual candidal vulvovaginitis.

Close-up of skin infection showing satellite pustules. Such lesions are sometimes seen in the axillae and in the submammary skin. (Courtesy Herman L. Gardner, Houston, Texas).

Acute vulvovaginal candidiasis – vulvar manifestations. Pruritus is the cardinal symptom. (Courtesy Herman L. Gardner, Houston, Texas)
Vulvovaginitis

- Women with uncontrolled diabetes, debilitating or those who are pregnant

B. Pathogenesis

Histologic analyses performed during the acute phase of infection do not reveal Candida albicans in the tissues. It is possible to experimentally reproduce some of the clinical characteristics of candidiasis by applying either dead Candida albicans or cellular extracts to the vulvovaginal mucosa. The clinical symptom seems to be a reaction to an allergen or to an endotoxin produced by the Candida.

Figure A. Diabetic vulvitis. Example of early case seen in diabetic patients with uncontrolled candidiasis.

Figure B. Example of advanced case in the patient with long-standing uncontrolled diabetes and untreated candidiasis. (Courtesy of Herman L. Gardner, Houston, Texas)

Vaginal candidiasis with thrush patches displayed. Only 15-20% of patients with the symptomatic infection display thrush patches. Many patients deny having a discharge. (Courtesy of Herman L. Gardner, Houston, Texas)

Updated!

C. Sources of Infection

With the intestinal tract as the reservoir of Candida, various species can be found in the stools, buccal cavity and vagina. In patients with clinical vaginal candidiasis, 75% of the cultures of feces are positive against 25% of those without a genital infection.

Candida is found in the buccal cavity of 20 to 30% of normal subjects. It is also found in the seminal fluid of the partners of patients suffering with a chronic infection.

Risk factors in the development of a vaginal sample reveals mycelia filaments, which proliferate from cellular closets, and spores; small circles represent a double contour.

The quickest and perhaps most accurate identification method is the microscopic examination of material mixed with a drop of a 10 to 20% potassium hydroxide solution. Pus globules, red blood corpuscles and vaginal cells containing keratin are rapidly dissolved. The conidia and filaments of the Candida remain.

The vaginal lesions can be cultured on Nickerson’s medium. Colonies of Candida albicans, stored at room temperature for 48 hours, are colored brown to black when the bismuth sulphite. For a more precise identifications of the species, various fermentation reactions of sugars are required.

E. Differential Diagnosis

Vulvovaginitis caused by Candida albicans can be distinguished from an infection caused by Trichomonas vaginalis or Haemophilus vaginalis by the malodorous smell of the two latter infections. Patients with chronic candidiasis, especially if diabetic, often show dermatologic signs of lichen planus or lichen sclerosus and atrophicus.

Due to scraping lesions, the epidermis can show signs of lichenification or present hyperkeratosis which resembles leukoplasia; however, these lesions are always red or brown in colour instead of white.

The chafing of two adjacent surfaces can provoke intertrigo, erythema followed by maceration due to perspiration. In this case, infection with Candida albicans is secondary. Eczema caused by contact with various toilet articles can also become superinfected with Candida albicans. The diagnosis is difficult because there is a cluster of vesicles and wheals around the main patch. Epicutaneous tests and mycologic research are required to solve the problem.

Candidal organisms in vaginal secretions. Hyphae and conidia as seen in physiological saline wet mount.
F. Treatment

Recommended Regimens

**Intravaginal Agents:**
- Butoconazole 2% cream 5 g intravaginally for 3 days **or**
- Butoconazole 2% cream 5 g (Butoconazole 1-sustained release), single intravaginal application **or**
- Clotrimazole 100 mg vaginal tablet for 7 days **or**
- Clotrimazole 100 mg vaginal tablet, two tablets for 3 days **or**
- Miconazole 2% cream 5 g intravaginally for 7 days **or**
- Miconazole 100 mg vaginal suppository, on suppository for 7 days **or**
- Miconazole 200 mg vaginal suppository, one suppository for 3 days **or**
- Miconazole 1,200 mg vaginal suppository, one suppository for 1 day **or**
- Nystatin 100,000-unit vaginal tablet, one tablet for 14 days **or**
- Tioconazole 6.5% ointment 5 g intravaginally in a single application **or**
- Terconazole 0.4% cream 5 g intravaginally for 3 days **or**
- Terconazole 80 mg vaginal suppository, one suppository for 3 days

**Oral Agent:**
- Fluconazole 150 mg oral tablet, one tablet in single dose

(Courtesy of CDC treatment guidelines, 2006)

IV. Parasitic Vaginitis

This group is mainly composed of vaginitis due to *Trichomonas vaginalis*, but for several years, amoebic vaginitis and vaginitis due to schistosomes have appeared and in the young girl, the consequences of oxyuriasis are often disregarded.

A. Vaginitis Due to *Trichomonas vaginalis*

Infections with *Trichomonas vaginalis* is the main cause of vulvovaginitis. *Trichomonas vaginalis* is a flagellated one-cell protozoon, which is oval in shape and the nucleus is eccentric.

Four flagella of the same length but being between one and two times the size of the organism itself stem from a blepharoblast as well as an axostyle, which traverses the protozoon from one pole to the other. This parasite lives in the urogenital sinus and provokes acute and chronic outbreaks of vaginitis.

A.1 Clinical description

The patient mainly complains of abundant leukorrhea, white, greenish, a burning sensation and sometimes dyspareunia. Vaginal discharge has unpleasant odor. Pruritus and burning can also exist, but are less intense than in mycotic infections.
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There is moderate edema and congestion of the vulva, sometimes combined with irritation of the inner side of the thighs. Vulvar erythema is usually limited to the labia minora and the vestibule.

The clinical diagnosis is facilitated by the presence of abundant, whitish, puffy leucorrhoea, which becomes foamy after gaseous fermentation of the aerogenic streptococcus. The vaginal mucus is often reddish-purple with dark red spots as a result of an erosion of the cervical and vaginal mucosa.

Half of the patients with this parasite do not present irritating symptoms.

A.2. Pathogenesis

Various hypotheses have been proposed to explain the cellular pathology induced by the infection, notably the production of a cytotoxic by the parasite and the existence of an enzymatic system capable of destroying the intercellular bridges.

A.3. Sources of infection

Infestation due to Trichomonas vaginalis is usually sexually transmitted. The Trichomonas can live for more than 24 hours in the urine and 6 hours on toilet seats in vaginal secretions. It slightly resists dessication.

The reservoir of the parasites is composed of the urethral and periurethral glands (of Skene), while Bartholin’s glands are more rarely infested. The parasite does not cross the cervical barrier and is never found in the uterus or tubes. It lives only briefly in the large intestine and can be distinguished from Trichomonas buccalis and Trichomonas hominis.

A.4. Biologic diagnosis

The parasite that can be seen by diluting the vaginal sample in one or two drops of physiologic liquid. It stands out because of its mobility, the numerous globules of pus and desquamative cells, which most often are parabasal in type. The absence of the parasite by direct examination does not exclude its presence; other investigations must be performed. An Ortho solution exists which turns pink when it is mixed with vaginal secretions containing Trichomonas. The vaginal cells become violet and the Trichomonas turns blue upon vital staining with brilliant cresyl blue at 1%. In smears colored by Papanicolaou, Trichomonas turns blue upon vital staining with brilliant cresyl blue at 1%. In smears colored by Papanicolaou, Trichomonas-gonocci, Trichomonas-innocuous germ and yeasts. These forms of vaginitis are frequently found in women who are carriers of chronic infections.

One might suspect a malignant lesion of the cervix in case of recurring mixed vaginitis combined with exocervicitis. Also, after the first anti-infectious treatment, cervico-vaginal samples must be taken for a Papanicolaou test.

Finally, emphysematous vaginitis, the etiology of which is unknown, very much resembles the clinical aspect of trichomoniasis. The vagina is spotted with small bubbles the size of a grain of rice, isolated or in groups.

Updated!

A.6. Treatment

Recommended Regimens:

- Metronidazole 2 g orally in a single dose
- Tinidazole 2 g orally in a single dose

Alternative Regimen:

- Metronidazole 500mg orally twice a day for 7 days

B. Amoebic Vaginitis

This type of vaginitis is very rare. Upon vaginal examination, liquid and purulent leucorrhoea is found with small ulcerations of the vaginal and cervical walls. The diagnosis is based on the demonstration of the parasite upon microscopic examination of the stools and leucorrhoea.

C. Vaginitis Due to Schistosomes

Schistosoma haematobium, also called Bilharzia haematobium, lives in infected water, penetrates through the skin and by the blood stream, invades various tissues where it provokes an inflammatory reaction. The lesions are predominantly found in hollow viscera, especially the bladder and the rectum, the genital tract, from the vulva to the ovaries.

The infection is chronic and sometimes can last the entire life. Vesical and intestinal symptoms are the prevailing symptoms. The genital infection is characterized by leucorrhoea, loss of blood, and dyspareunia. It promotes sterility because the antibodies are spermatotoxic.

On the cervix, the infection appears as an erosion or leukoplakia, which often suggests a neoplastic lesion. A histologic analysis shows a severe inflammatory reaction, each cell being surrounded by giant cells, epithelial cells,
lymphocytes and eosinophils.

D. Vaginal Oxyuriasis

*Oxyuris vermicularis* and Enterobius lay their eggs during the night around the anus and in the vagina, which do not provoke a discharge but cause itching.

V. Microbic Vulvovaginitis

Today, there is a new outbreak of Gonococcal vaginitis due to haemophilus and vaginitis due to innocuous germs.

A. Gonococcal Vaginitis

Gonorrhea is caused by diplococcus *Neisseria gonorrhoea*, which must be differentiated from other saprophytic non-gonococcal Neisseria of the vagina.

A.1. Clinical Description

There is painful vulvar edema-accompanied by greenish leucorrhoea, which appear 2 to 7 days after the sexual act. This latency period increases to 4 to 8 weeks when the Neisseria stock is only slightly virulent and the host has a high resistance. When cleaned of the greenish purulent leucorrhoea the mucus membrane can be seen to be red and oedematous; unfolding the vulva reveals that the orifices of Skene’s and Bartholin’s glands are greatly congested. Urethritis is also present, provoking dysuria and even urine retention. Purulent exudates provoked by compression of the urethra. About 3/4 of the patients suffering from this infection are quasi-asymptomatic.

A.2. Pathogenesis

Gonococcus mainly affects the glandular structures. In the acute phase, hyperemia, edema, and an infiltration of the mucous membrane and submucosa by poly-nuclears can be seen.

The destruction of the mucous membrane varies, but it can suffice to promote stenosis of the excretory canals. This is also secondary to the pericanalicular fibrous retraction. The vulvar skin may present an allergic type of erythematous reaction to purulent exudates of the glands of the lower genital tract.

A.3. Sources of infection

The infection is mainly contracted through sexual relations. However, newborns and children could be infected through the maternal pelvic cavity during delivery or by their underwear.

A.4. Biologic diagnosis

During the acute phase, it is possible to collect secretions and to stain them by the gram method. Gonococcus is an intra and extracellular coccus of 0.6 to 0.8 microns appearing in pairs; it is gram-negative.

The diagnosis can only be established by a bacteriologic examination of the exudates of the cervix, urethra and Skene’s and Bartholin’s glands. The gonococcus is very fragile and the sample must be transported and kept in Stewart’s medium for 24 to 48 hours at a temperature of 4°C. Among the numerous culture mediums, chocolate agar and Thayer-Martin, in an atmosphere of 3 to 10% carbon dioxide at 35-36°C are the best. In the chronic form of the illness, a valid technique is the search for antibodies by fluorescence.

A.5 Differential diagnosis

Gonorrhoea can accompany all types of vaginal infection especially trichomoniasis. Concomitant syphilis must not be neglected.

**Figure A**

Gonorrhoea. Gram-stained smear-dependable when a purulent urethritis or cervicitis is present but unreliable as a screening procedure.

**Figure B**

Thayer-Martin culture plate showing typical colonies and positive oxidase reaction. This test is 90% reliable. (Courtesy of Herman L. Gardner, Houston, Texas).

B. Vaginitis Due *Haemophilus vaginalis*

This vaginal infection is still underestimated in our region probably due to the difficulties in culturing this gram-negative bacillus.

Updated!

It is a polymicrobial clinical syndrome resulting from replacement of the normal H2O2-producing Lactobacilus sp. In the vagina with high concentrations of anaerobic bacteria (e.g., Prevotella sp. and Mobiluncus sp.) Gardnerella vaginalis and Mycoplasma hominis.

B.1. Clinical description

Clinical criteria require three of the following symptoms or signs:

- Homogenous, thin, white vaginal discharge that smoothly coats the vaginal walls;
- Presence of clue cells on microscopic examination;
- pH of vaginal fluid >4.5; and
- A fishy odor of vaginal discharge before or after addition of 10% KOH (i.e., the whiff test).

Irritation, burning sensation and an increase of the vaginal discharge are common symptoms of this infection. The malodorous discharge, less abundant than trichomoniasis, is viscous, either grey, white or greenish-grey in color.

B.2. Pathogenesis

*Haemophilus vaginalis* is less aggressive and virulent than gonococcus and trichomonas. It can accompany gonococcus and trichomonas.

B.3. Sources of infection

The most frequent means of transmission is the venereal contact. The germ lives in the male urethra and its multi-
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Plication depends on the supply of the urethral epithelium and the frequency of the urinary flow. It is found in 90% of the partners of the infected patients without them being inconvenienced by it.

**B.4. Biological diagnosis**

*Haemophilus vaginalis*, which can be detected through vaginal smear, reveals a cytoplasmic reaction and the appearance of granulas which are distributed uniformly on the cellular surface of the vaginal epithelium. These cells or "clue cells" are characteristic of infection with *H. vaginalis*.

The bacilli are seen as small rods, gram-negative intra-or extracellular. Lactobacillus is often absent.

**Updated!**

**B.5. Treatment:**

**Recommended regimens:**

- Metronidazole 500 mg orally twice a day for 7 days or
- Metronidazole gel 0.75%, one full applicator (5g) intravaginally, once a day for 5 days or
- Clindamycin cream 2%, one full applicator (5g), intravaginally at bedtime for 7 days

**Alternative regimens:**

- Clindamycin 300 mg orally twice a day for 7 days or
- Clindamycin ovules 100 mg intravaginally, once at bedtime for 3 days.

**C. Vaginitis Due to Innocuous Germs**

This can include all pyogenic germs, staphylococcus, streptococcus, Proteus, enterococcus, mycoplasma, colibacillus, diphereral bacilli, *Listeria monocytogenes*, etc. They can become pathogenic following a trauma, intercurrent infectious illness, the presence of a foreign body especially during childhood or an exocervicitis.

The leucorrhoea is greenish and yellow and purulent. The vulvo-perineal irritation and dyspareunia are less constant than in the preceding infections. The vaginal pH is higher than 6. Infections due to mycoplasma or *Listeria monocytogenes* can cause sterility, miscarriages and fetal death during the 2nd trimester of pregnancy.

**VI. Viral Vulvovaginitis**

There are three most frequent viral affections: Condyloma acuminatum, herpes genitalis and molluscum contagiosum.

**A. Condyloma acuminatum**

Condyloma acuminatum and the common wart present a similar structure. Although it has not yet been proven, the same virus might be the cause of the two afflictions.

**A.1. Clinical description**

The vegetations usually grow on the vulva, the ves- tibule and in the folds of the labia majora. They are rarely found on the perineal skin, mons veneris, anal edge and less exceptionally on the vaginal and cervi- cal mucosa. The vegetations are rather inconspicuous in the beginning of the affection and cause the patient little discomfort. These vegetations vary in color from pink to brown. Their growth then provokes tumours often luxuriant, particularly during pregnancy. These lesions can become ulcerous and infected. The patient experiences a burning sensation.

![Clue cells of *H. vaginalis* vaginitis. Normal epithelial cell upper right. Pus cells are few and no lactobacilli are present. (Courtesy of Herman L. Gardner, Houston, Texas).](image)

**A. Condylomata acuminata. Classical case. (Courtesy of Herman L. Gardner, Houston, Texas)**

**B. Perianal warts. Lesions also present in anal canal. (Courtesy of Herman L. Gardner, Houston, Texas)**

**A.2. Pathogenesis**

The acuminate warts develop during the period of repro-duction and are exceptional before puberty and during postmenopause. They are most probably transmitted through sexual relations, but a hormonal condition as that of pregnancy can also come into play. The affection is more frequently found in carriers of gonorrhoea, trichomoniasis and candidiasis.

**A.3. Differential diagnosis**

The very evocative clinical aspect should not rule out the possibility of vulvar cancer. The lesions, which are suspected to be neoplastic, are fluorescent when the vulva is examined. In addition, the questionable lesions remain blue after washing the vulva with a solution of Toluidine blue at 1 %, followed, after 2 minutes, by a washing with 1 % acetic acid.

![Condylomata acuminata, extensive in this pregnant pa- tient. (Courtesy of Herman L. Gardner, Houston, Texas).](image)
Condylomata acuminata. Lesions in vagina of a pregnant woman. Vaginal warts are common even in the non-pregnant patient. (Courtesy of Herman L. Gardner, Houston, Texas)

Condylomata encircling cervix. (Courtesy of Herman L. Gardner, Houston, Texas)

Updated!

Recommended Regimens for External Genital Warts

**Patient-Applied:**
- **Podofilox** 0.5% solution or gel. Apply with a cotton swab, or with a finger, twice a day for 3 days, followed by 4 days of no therapy. Cycle may be repeated, as necessary, for up to four cycles.
- **Imiquimod 5% cream.** Apply once daily at bedtime, three times a week for up to 16 weeks. The treatment area should be washed with soap and water 6-10 hours after the application.

**Provider-Administered:**
- **Cryotherapy** with liquid nitrogen or cryoprobe. Repeat applications every 1-2 weeks.
- **Podophyllin resin 10%-25% in a compound tincture of benzoin.** Repeat weekly, if necessary.
- **Trichloroacetic acid (TCA) or Bichloroacetic acid (BCA) 80%-90%**, 
- **Surgical removal** either by anogenital scissor excision, tangential shave excision, curettage, or electro-surgery.

**Alternative Regimens:**
- Intralesional interferon
- Laser surgery

Recommended Regimens for Vaginal Warts

- Cryotherapy with liquid nitrogen. The use of a cryoprobe is not recommended because of the risk for vaginal perforation and fistula formation.
- Trichloroacetic acid (TCA) or Bichloroacetic acid (BCA) 80%-90% applied to warts

Recommended Regimens for Urethral Meatus Warts

- Cryotherapy with liquid nitrogen
- Podophyllin 10%-25% in compound tincture of benzoin

Recommended Regimens for Anal Warts

- Cryotherapy with liquid nitrogen
- Trichloroacetic acid (TCA) or Bichloroacetic acid (BCA) 80%-90% applied to warts
- Surgical removal

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**H.P.V. Types of Relative Cancer Risk**

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<th>CANCER RISK</th>
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<td>INTERMEDIATE</td>
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<td>16/18, 45, 56</td>
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**Reference:**

**B. Herpes Genitalis**

Herpes Genitalis is a chronic, life long viral infection. Two types of HSV have been identified HSV-1 characterized by buccal-facial manifestations and HSV-2 has genitourinary manifestations and a venereal transmission. Herpes of the newborn is almost always of the latter type, resulting from herpes genitalis of the mother. The importance of this infection has increased because of its role in the etiology of certain types of cancer of the cervix.

**B.1. Clinical description**

The symptoms range from cutaneous hyperaesthesia, to pruritus and perineal burning. Secondary leucorrhoea usually occurs and is intense. Urination is often painful; the retention of urine is exceptional.

The primary lesion sometimes lasts from 3 to 6 weeks, while these symptoms, which periodically reappear, last between 7 and 10 days. The primary infection can be accompanied by fever, headache, indigestion, anorexia. The lesions are usually found near the vestibule, labia and in the periclitoridean region. The cervix also is possibly infected.

Ulceration occurs and lasts between 7 and 10 days. The base if erythematous and the ulcer is covered with yellowish exudates. Vesicles and ulcers can unite and become superinfection. However, the vesicles can persist and dry on the labia majora, which are less subject to maceration. The vesicles are less typical on the cervical and vaginal mucosa. They resemble a deposit of mucus. A superinfection can provoke considerable necrosis, which is often confused with a cancerous lesion.

**B.2. Biologic diagnosis**

Herpes genitialis must also be suspected when vulvovaginal vesicles or ulcerations are observed. The clinical diagnosis is easily made during the primary infection, but difficult during relapses.

Cytologic examination shows giant multinucleate and very vacuolated cells. The easiest manipulation is the serologic method with measurement of the antibody treatment.
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B.3. Differential diagnosis

Herpes zoster is always unilateral and cannot be confused with herpes genitalis

Figure A. Histologic section through a primary lesion showing formation of multinucleate giant cells.

Figure B. Cytologic smear from recurrent lesions showing diagnostic epithelial giant cells. Condylomata acuminate, extensive in this pregnant patient. (Courtesy of Herman L. Gardner, Houston, Texas)

Updated!

B.4. Treatment

- HPV Vaccines
- Prophylactic HPV L1 VLP Vaccines

<table>
<thead>
<tr>
<th>QUADRIVALENT VACCINE</th>
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<tr>
<td>Vaccine type</td>
<td>HPV 6/11/16/18</td>
</tr>
<tr>
<td>Schedule</td>
<td>0.5 mL IM x 3 separate doses</td>
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<td>0.5 mL IM x 3 separate doses</td>
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First Clinical episode of Genital Herpes

Recommended Regimens

- Acyclovir 400 mg orally three times a day for 7-10 days
- Acyclovir 200 mg orally five times a day for 7-10 days
- Famciclovir 250 mg orally three times a day for 7-10 days
- Valacyclovir 1 g twice a day for 7-10 days

Suppressive Therapy for Recurrent Genital Herpes

Recommended Regimens

- Acyclovir 400 mg orally twice a day
- Famciclovir 250 mg orally twice a day
- Valacyclovir 500 mg orally once a day
- Valacyclovir 1.0 g orally once a day

Episodic Therapy for Recurrent Genital Herpes

Recommended Regimens

- Acyclovir 400 mg orally three times a day for 5 days
- Acyclovir 800 mg orally twice a day for 5 days
- Acyclovir 800 mg orally three times a day for 2 days
- Famciclovir 125 mg orally twice daily for 5 days
- Famciclovir 1000 mg orally twice daily for 1 day
- Valacyclovir 500 mg orally twice a day for 3 days
- Valacyclovir 1.0 g orally once a day for 5 days

C. Molluscum contagiosum

The molluscum contagiosum virus is one of the biggest known viruses. The affliction is often found in the child, where it often becomes epidemic

C.1. Clinical description

The contagious affection is characterized by the presence of small papulous formations, rounded, salient—firm in consistency, grayish-white in colour, with a navel-shaped depression in the middle, pruriginous, multiple and sometimes isolated. These lesions are found in the genital region, and also on the face and arms. They multiply rapidly.

C.2. Histologic diagnosis

The acanthotic epithelium penetrates deeply in the dermis to form characteristic lobules. The epithelial cell is greatly eosinophilic and contains large cytoplasmic inclusions, which crush the nucleus to form an eosinophil mass surrounded by keratohyaline granulas. These masses join together and make up a corpuscle of molluscum, which desquamates when it reaches the epithelial surface. (Courtesy of Herman L. Gardner, Houston, Texas)

C.3. Differential diagnosis

Smaller eosinophilic granules are found in the stratum granulosum of the hyperkeratotic wart.

Dome-shaped, papular lesions with diagnostic umbilications. (Courtesy of Herman L. Gardner, Houston, Texas)

Molluscum contagiosum—epithelial cells containing cytoplasmic inclusion bodies. (Courtesy of Herman L. Gardner, Houston, Texas)

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C. Molluscum contagiosum

The molluscum contagiosum virus is one of the biggest
Vulvovaginitis

Without oestrogens, the vulvovaginal mucosa is reduced to the deep and parabasal intermediary level. The natural defense mechanisms of the vagina have greatly decreased due to the fact that the glycogen has disappeared, there is no more lactic acid and the pH is alkaline. The mucosa is easily altered by pyogenic germs, especially colibacillus, enterococcus and staphylococcus.

C. Biologic Diagnosis

Upon application of Lugol on the cervix and after having been diluted in a physiologic solution, the vaginal discharge was examined under the microscope and showed the presence of intermediary malpighian bodies with voluminous nuclei and parabasal cells.

D. Differential Diagnosis

The atypical cells can resemble cancer cells. They disappear after estrogenic reimpregnation of the mucosa. The pinkish discharge may be due to an intra-uterine neoplastonic lesion.

Updated!

E. Treatment

- Estrogenic

VIII. Chlamydia Trachomatis

- Most common STD in developed countries.
- True Virus
- One of the principal causes of blindness
- 8% - P1D - Infection of unborn infant
- Obligate intracellular parasite but is nevertheless classified as a bacterium
- Causes selective phagocytosis in host cells.
- Development cycle at 30 hours.
- 1.1, 1.2, L3 = L.G. Venereum
- A. B1, Ba, C, D & K = Trachoma, conjunctivitis and common genital infections.

Incidence

- 2% in vagina of women without sexual contact.
- 17% M Hominis
- 75% Urea plasma

Asymptomatic Chlamydia

10-20% of infected males, 80% of infected females

Repeated and Untreated Infections

- PID
- Infertility
- Chronic Abdominal pain
- Ectopic pregnancy increase by 10%
- 50% of newborn - conjunctivitis
- 20% of chlamydia pneumonia

Updated!

IX. Mycoplasma and Urea Plasma

- First reported in 1937
- Smallest known free-living organisms
- Not larger than 300 NM in diameter
- 1974 - species - metabolize
- Urea = Urea plasma urealyticum

Detection and Diagnosis of Chlamydia

- 20 - 20% of Males and 40 - 45% of Females with G.C.
- Tissue culture
- DFA - Direct - Smear fluorescent antibody
- Eliza — Enzyme linked Immune-absorbent assay

M. Hominis

- Acute PID
- Post-partum and post-abortal fever
- Arthritis after childbirth

Updated!

FEMININE HYGIENE ADVISORY:

Intimate area care

- Wash hands thoroughly before and after handling genital area
- Wash only the perineal area and use mild soap and water
- Rinsing with water should be directed downwards without touching the anal region
- Change underwear every day
- Wear underwear with a cotton crotch to help pull extra moisture away from the body
- Observe proper technique on genital care particularly during menstrual days
- Change pads every 4 to 6 hours when your are having a menstrual period to avoid irritation and lower the risk of infection
- Sleep in a loose fitting pajama bottoms
- Do not use tampons, pads soaps or douches that are perfumed
Recommended Therapeutics

The following index lists therapeutic classifications as recommended by the treatment guideline. For the prescriber’s reference, available drugs are listed under each therapeutic class. For drug information, please refer to the Philippine Drug Directory System (PPD, PPDr, PPD Text, PPD Tabs).

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<td>Abbott - Metronidazole</td>
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<td>Drugmaker’s Biotech Metronidazole</td>
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<td>Zmax One Dose</td>
<td>Flagyl</td>
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