LESSON TITLE:
CARE OF PATIENTS WITH ALTERATION/PROBLEMS IN INFECTIOUS AND INFLAMMATORY DISORDERS: STD, HIV and AIDS

Sexually Transmitted Diseases

Sexually transmitted diseases (STDs) — or sexually transmitted infections (STIs) — are generally acquired by sexual contact. The organisms (bacteria, viruses or parasites) that cause sexually transmitted diseases may pass from person to person in blood, semen, or vaginal and other bodily fluids

Infection	Cause	Clinical Manifestation	Pathophysiology	Management		
				Diagnostics	**Treatment**	**Medication**
Candidiasis	Candida albicans, glabrata, or tropicalis	reddish irritation White, cheese like discharge clinging to epithelium	Inflammation of vaginal epithelium, producing itching	Diagnosis is made by microscopic identification of spores and hyphae on a glass slide prepared from a discharge specimen mixed with potassium hydroxide. With candidiasis, the pH is 4.5 or less.	Review other causative factors (e.g., antibiotic therapy, nylon underwear, tight clothing, pregnancy, oral contraceptives). Assess for diabetes and HIV infection in patients with recurrent monilia.	Eradicate the fungus by administering an antifungal agent. Frequently used brand names of vaginal creams and suppositories are Monistat, Femstat, Terazol, and Gyne-Lotrimin
Trichomonas vaginalis vaginitis (STD)	Trichomonas vaginalis	Frothy yellow-white or yellow-green vaginal discharge	Inflammation of vaginal epithelium, producing burning and itching	Flagellated, motile trichomonads on wet mount Vaginal pH > 4.5 Diagnosis confirmed by microscopy Other FDA approved tests: OSOM Trichomonas Rapid Test Affirm VP III	Relieve inflammation, restore acidity, and reestablish normal bacterial flora;	provide oral metronidazole for patient and partner.
Cervicitis: Acute and Chronic	Chlamydia Gonococcus Streptococcus Many pathogenic bacteria	Profuse purulent discharge Backache Urinary frequency and urgency	Inflammation of vaginal epithelium, producing burning and itching	Direct immunofluorescence assays (DFA) Enzyme immunoassay (EIA)	Determine the cause: perform cytologic examination of cervical smear and appropriate cultures. Eradicate other causes. Evaluate and treat sexual partners Avoid sex for seven days after completion of treatment	Eradicate the gonococcal organism, if present: penicillin (as directed) or spectinomycin or tetracycline, if patient is allergic to penicillin. Tetracycline, doxycycline (Vibramycin) to eradicate chlamydia.
Herpes Simplex	Herpes Simplex Virus	Appearance of painful vesicles in clusters on an erythematous base	Virus must come in contact with mucosal surfaces or abraded skin for infection to be initiated. With viral replication at the site of primary	Swab test Blood test Lumbar Puncture	Lesions may be bathed in mild soap and water Sitz baths may provide some relief Sex partners	Antivirals

		Tingling/itching of skin	infection, either an intact virion or, more simply, the capsid is transported retrograde by neurons to the dorsal root ganglia where, after another round of viral replication, latency is established		may benefit from evaluation and counseling	
Syphilis	spirochaete Treponema pallidum	Primary chancre formation in the genital area fever, malaise, generalized lymphadenopathy, and patchy alopecia	Begins as papule and erodes into painless ulcer with a hard edge and clean base	Venereal Disease Research Laboratory (VDRL) Rapid Plasma Reagent (RPR) Fluorescent Treponemal Antibody Absorption (FTA-ABS) Micro hemagglutination-Treponema pallidum (MHA-TP)	Watch out and manage Jarisch-Herxheimer reaction Avoid sexual contact with new partners until the treatment is completed	Benzathine penicillin Penicillin G
Gonorrhea	N. gonorrhoeae -gram negative diplococci	Frequently asymptomatic Vaginal discharge Abnormal uterine bleeding Dysuria Mucopurulent cervicitis Lower abdominal pain	utilizes glucose, but not sucrose, maltose, or lactose. It can infect mucus-secreting epithelial cells in both men and women.	Clinical exam Cervical culture Polymerase chain reaction (PCR) or ligase chain reaction (LCR) Gram stain–polymorphonucleocytes with gram negative intracellular diplococci	Evaluate and treat all sexual partners	Intramuscular Ceftriaxone For pregnant women only: Ceftriaxone single dose but substitute Quinolones with Erythromycin Do not treat with Quinolones or Tetracyclines
Human papillomavirus (HPV)	Human papillomavirus (HPV)	Genital warts Common warts Plantar warts Flat warts	The most common strains of HPV, 6 and 11, usually cause condylomata (warty growths) on the vulva.	Vinegar (acetic acid) solution test. Pap smear DNA test	Surgical management of warts: Freezing with liquid nitrogen (cryotherapy) Burning with an electrical current (electrocautery) Surgical removal Laser surgery	Medications to eliminate warts are typically applied directly to the lesion and usually take many applications before they're successful. Salicylic acid. Imiquimod Podofilox. Trichloroacetic acid

					Have HPV vaccine	

Nursing Care Plan

Nursing Diagnosis	Intervention	Evaluation
Risk for Infection	✓ Maintain or teach asepsis for dressing changes and wound care, peripheral IV and central venous management, and catheter care and handling. ✓ Wash hands and teach patient and SO to wash hands before contact with patients and between procedures with the patient. Instances when to wash hands: — Before putting on gloves and after taking them off. — Before and after touching a patient, before handling an invasive device (foley catheter, IV catheter, and so on) regardless of whether or not gloves are used. — After contact with body fluids or excretions, mucous membranes, nonintact skin, or wound dressings. — If moving from contaminated body site to another site during the care of the same individual. — After contact with inanimate surfaces and objects in the immediate vicinity of the patient. — After removing sterile or nonsterile gloves. — Before handling medications or preparing food. ✓ Encourage intake of protein-rich and calorie-rich foods. ✓ Encourage fluid intake of 2,000 to 3,000 mL of water per day, unless contraindicated.	✓ Patient remains free of infection, as evidenced by normal vital signs and absence of signs and symptoms of infection. ✓ Early recognition of infection to allow for prompt treatment. ✓ Patient will demonstrate meticulous hand washing technique.
Acute Pain related to genital lesions	✓ Suggest use of non-pharmacological techniques as appropriate. ✓ Encourage increased oral fluid intake (2-3 liters if no contraindications). ✓ Encouraged the use of a sitz bath. ✓ Instruct to avoid coffee, tea, alcohol, and sodas. ✓ Apply a heating pad to the suprapubic area or lower back. ✓ Encouraged the use of analgesic (e.g., acetaminophen) or antispasmodics (e.g., phenazopyridine) as prescribed	✓ Client will use pharmacological and nonpharmacological pain relief strategies. ✓ Client will report satisfactory pain control at a level less than 3 to 4 on a scale of 0 to 10.

Add ons:

Candidiasis

Candidiasis is a fungal infection caused by *Candida* species, predominantly *Candida albicans*. It can manifest in various forms, including oral thrush, vaginal yeast infections, and systemic infections. This discussion covers multiple aspects of candidiasis relevant to nurses and doctors.

Causative Agent

- **Causative Agent**: *Candida albicans* is the most common fungal pathogen, but other species like *Candida glabrata*, *Candida tropicalis*, and *Candida krusei* can also cause infection.
- **Rationale**: *Candida albicans* is a commensal organism, meaning it normally resides on mucosal surfaces of the human body (oral cavity, vagina, gut, and skin) without causing harm. Under certain conditions (e.g., immunosuppression, disruption of the normal flora), it can become pathogenic, leading to overgrowth

and infection.

Anatomy and Physiology

- **Anatomy**: The primary sites affected by *Candida* are mucosal surfaces (oral cavity, esophagus, gastrointestinal tract, genital tract) and the skin.
- **Physiology**: *Candida* is part of the normal flora, and its growth is typically kept in check by the immune system and the microbiota. Disruption of these factors leads to overgrowth.
- **Rationale**: The balance between *Candida* and host defenses is critical. Disruption by antibiotics, immunosuppressive therapy, diabetes, or hormonal changes (pregnancy) can alter the mucosal environment, enabling infection.

Pathophysiology

- **Pathophysiology**: In candidiasis, *Candida* transforms from a yeast form into an invasive hyphal form. The hyphal form penetrates mucosal tissues, causing inflammation and tissue damage. It can disseminate into the bloodstream, leading to systemic candidiasis.
- **Rationale**: The ability of *Candida* to switch forms is crucial to its pathogenicity. The hyphal form allows for deeper tissue invasion, leading to both localized and systemic infections in immunocompromised individuals.

Incubation Period

- **Incubation Period**: The incubation period varies depending on the type of candidiasis. Superficial infections can develop within a few days, whereas systemic candidiasis may take longer, depending on the immune status.
- **Rationale**: The length of the incubation period depends on the immune response of the host. Immunocompromised patients may experience a more rapid onset of symptoms.

Mode of Transmission

- **Mode of Transmission**: *Candida* is not typically transmitted from person to person. Instead, candidiasis results from endogenous overgrowth. However, in rare cases, it can be transmitted through contaminated medical devices (e.g., catheters) or during childbirth (neonatal candidiasis).
- **Rationale**: Since *Candida* is part of the normal flora, transmission is usually not an issue. Invasive procedures that breach sterile

barriers can introduce *Candida* into the bloodstream or other sterile sites.

Risk Factors

- **Risk Factors**:
  - Immunosuppression (e.g., HIV, cancer therapy)
  - Diabetes mellitus
  - Use of broad-spectrum antibiotics or corticosteroids
  - Pregnancy
  - Use of indwelling devices (e.g., catheters)
  - Poor oral hygiene or use of dentures
  - Disruption of mucosal barriers (e.g., chemotherapy)
- **Rationale**: These conditions either weaken the immune system, disrupt the balance of normal flora, or provide a portal for infection.

Signs and Symptoms

- **Signs and Symptoms**:
  - **Oral candidiasis (thrush)**: White plaques on the tongue and mucous membranes, painful swallowing.
  - **Vulvovaginal candidiasis**: Itching, burning, white "cottage cheese" discharge, redness, and swelling.
  - **Cutaneous candidiasis**: Red, itchy skin rashes, often in skin folds (e.g., under breasts, groin).
  - **Systemic candidiasis**: Fever, chills, sepsis, organ failure (liver, spleen, kidneys).
- **Rationale**: The symptoms reflect the localized inflammatory response caused by *Candida* overgrowth and tissue invasion. In systemic cases, dissemination can lead to multi-organ involvement.

Assessment

History and Physical Examination:

- **History**:
  - Ask about recent use of antibiotics, corticosteroids, or immunosuppressants.
  - Evaluate for conditions like diabetes or HIV.
  - Ask about oral hygiene practices, denture use, and symptoms like vaginal itching or discharge.
- **Physical Examination**:
  - Inspect oral mucosa for white plaques or redness.

Examine the skin for erythema, rashes, and fissures, especially in skin folds.
    - Inspect vaginal discharge and perform a pelvic exam for vulvovaginal candidiasis.
    - Assess for signs of systemic infection (fever, hypotension, multi-organ failure).
- **Rationale**: Proper history and examination help identify potential predisposing factors and confirm the diagnosis through characteristic findings.

Laboratory and Diagnostic Procedures

- **Laboratory Tests**:
    - **KOH preparation**: Scraping from affected areas, treated with potassium hydroxide, shows yeast and pseudohyphae under a microscope.
    - **Culture**: A swab or tissue biopsy can be cultured to identify *Candida* species.
    - **Blood cultures**: In systemic candidiasis, blood cultures are positive for *Candida*.
    - **Complete Blood Count (CBC)**: Look for leukocytosis in systemic infections.
- **Diagnostic Imaging**:
    - **Endoscopy**: For esophageal candidiasis, endoscopy can reveal white plaques in the esophagus.
- **Rationale**: Microscopy and culture confirm the presence of *Candida*. Blood cultures are essential for detecting systemic involvement.

Medical Management

- **Topical Antifungals**: Clotrimazole, nystatin for oral and cutaneous candidiasis.
- **Oral Antifungals**: Fluconazole for oral, esophageal, and vaginal candidiasis.
- **Intravenous Antifungals**: Amphotericin B or echinocandins (e.g., caspofungin) for systemic candidiasis.
- **Rationale**: Treatment depends on the severity and location of the infection. Topical agents are effective for localized infections, while systemic agents are needed for invasive candidiasis.

Procedures and Surgery

- **Central Line Removal**: In cases of catheter-related candidemia, removing the infected catheter is essential. - **Surgical Drainage**:

For abscesses or collections caused by *Candida*, surgical drainage may be required.

- **Rationale**: Removing the source of infection (e.g., catheters or abscesses) is critical to preventing further dissemination.

Nursing Management

Assessment:

- Monitor for signs of infection (fever, rash, discharge). • Assess mucosal membranes and skin folds regularly. • Check for side effects of antifungal medications. • **Rationale**: Early detection and monitoring for complications can help prevent progression to systemic infection.

Intervention:

- Administer prescribed antifungal medications on time. • Educate the patient on proper hygiene (e.g., oral care, keeping skin folds dry).
- For vaginal candidiasis, recommend wearing loose-fitting, cotton underwear.
- **Rationale**: Proper medication administration and hygiene are crucial to managing infection and preventing recurrence. **Health Teaching:**
- Teach patients to avoid excessive use of antibiotics and to maintain good hygiene.
- Instruct patients with diabetes to keep blood sugar levels under control.
- Educate on completing the full course of antifungals to prevent resistance.
- **Rationale**: Educating the patient empowers them to manage risk factors and prevent future infections.

Nursing Goal

- **Goal**: Prevent the progression of localized candidiasis to systemic infection and promote complete resolution of symptoms.
- **Rationale**: Prompt and appropriate treatment of localized infections reduces the risk of systemic complications.

Nursing Priority

- **Priority**: Early identification and treatment of infection, especially in immunocompromised patients, to prevent systemic dissemination.
- **Rationale**: Immunocompromised patients are at high risk for life-threatening systemic candidiasis.

Nursing Actions

- **Action**: Perform thorough skin and mucosal assessments and report any signs of worsening infection or systemic involvement.
- **Rationale**: Regular assessment helps detect early signs of complication, allowing for timely intervention.

Prevention

- **Prevention**:
  - Maintain proper hygiene.
  - Avoid overuse of antibiotics.
  - Control chronic diseases like diabetes.
  - Use sterile techniques for invasive devices like catheters.
- **Rationale**: Preventive measures reduce the risk of infection by maintaining the body's natural defenses and minimizing the introduction of pathogens.

Trichomonas Vaginalis Vaginitis (STD)

Trichomonas vaginalis vaginitis, commonly known as "trichomoniasis," is a sexually transmitted infection (STI) caused by a protozoan parasite. It affects both men and women but is more commonly symptomatic in women.

Causative Agent

- **Causative organism**: *Trichomonas vaginalis* is a flagellated protozoan.
- **Rationale**: This organism specifically infects the genitourinary tract, where it attaches to the epithelial cells and causes inflammation. Its flagella allow motility, contributing to the invasion and irritation of the mucosal lining.

Anatomy and Physiology

- **Female genital tract anatomy**: The vagina, cervix, and urethra are the primary sites of infection.
- **Rationale**: The moist, warm environment of the vagina is ideal for the growth and multiplication of *Trichomonas vaginalis*. The organism thrives in this low-pH environment, and the cervix and urethra are highly susceptible to infection because of the epithelial cell lining, which can be invaded by the protozoan.

Pathophysiology

- **Invasion**: *Trichomonas vaginalis* adheres to the squamous epithelial cells of the vagina, urethra, and cervix.
- **Inflammation**: The protozoa secrete cytotoxic substances that cause cellular damage and inflammation.
- **Discharge**: The infection results in increased vaginal discharge, which can be frothy, yellow-green, and malodorous due to the metabolic byproducts of the protozoa and the immune response of the host.
- **Rationale**: The organism triggers an immune response, leading to inflammation, discomfort, and characteristic discharge. Inflammation is the body's response to the invasion of the epithelial lining.

Incubation Period

- **Range**: 5 to 28 days.
- **Rationale**: The variation in incubation time depends on the organism's load, the host's immune status, and environmental factors within the genitourinary tract.

Mode of Transmission

- **Primarily sexual**: *Trichomonas vaginalis* is transmitted via vaginal, oral, or anal intercourse.
- **Rationale**: The organism cannot survive outside the human body for long periods and requires close physical contact to spread. Sexual transmission is facilitated by the direct contact of genital mucosa.

Risk Factors

1. **Unprotected sexual intercourse**: Increases the risk of transmission.
    - **Rationale**: Lack of barrier protection facilitates direct contact between mucosal surfaces.
2. **Multiple sexual partners**: Increases the probability of encountering an infected individual.
    - **Rationale**: The more partners, the higher the chance of exposure to the protozoa.
3. **Other STDs**: Co-infection with other sexually transmitted infections like HIV.
    - **Rationale**: Weakened mucosal immunity due to another infection makes individuals more susceptible.
4. **Poor personal hygiene**: May increase the growth of the organism in the genital area.

- **Rationale**: The moist, unclean environment fosters the proliferation of pathogens.

Signs and Symptoms

- **Females**:
  - Vaginal discharge: Frothy, yellow-green, malodorous.
    - **Rationale**: Due to inflammation and increased secretion of fluids.
  - Vulvar irritation and pruritus.
    - **Rationale**: Resulting from the irritation of mucosal surfaces.
  - Dysuria (painful urination).
    - **Rationale**: Due to inflammation of the urethra.
  - Dyspareunia (painful intercourse).
    - **Rationale**: Inflammation of the vaginal mucosa makes intercourse painful.
- **Males** (often asymptomatic but may have):
  - Urethral discharge.
    - **Rationale**: Inflammation of the urethral lining due to protozoa.
  - Dysuria.
    - **Rationale**: Urethral inflammation can cause discomfort during urination.

Assessment: History and Physical Examination

- **History**:
  - Sexual history: Multiple partners, unprotected sex.
    - **Rationale**: Identifying risk factors for exposure to *Trichomonas vaginalis*.
  - Previous STDs.
    - **Rationale**: History of other STIs increases susceptibility.
- **Physical examination**:
  - **Females**: Vaginal examination may show erythema, edema, and frothy discharge from the vagina or cervix.
  - **Males**: Less commonly, urethral discharge may be present.
  - **Rationale**: Physical findings help in identifying the site of infection and the extent of inflammation.

Laboratory and Diagnostic Procedures

1. **Microscopy of vaginal discharge**:

- **Wet mount preparation**: Identification of motile *Trichomonas vaginalis* under a microscope.
- **Rationale**: Direct visualization of the organism is a quick and cost-effective diagnostic tool.

2. **Nucleic Acid Amplification Test (NAAT)**:
   - **Rationale**: High sensitivity and specificity make it the gold standard for diagnosing trichomoniasis.
3. **Culture of vaginal/urethral discharge**:
   - **Rationale**: Less commonly used but effective in cases of unclear diagnosis.
4. **pH of vaginal secretions**:
   - Elevated vaginal pH (>4.5) is indicative of infection.
   - **Rationale**: *Trichomonas* increases the alkalinity of vaginal fluids.

Medical Management

- **Metronidazole or Tinidazole (first-line treatment)**:
  - Dose: 2 grams orally in a single dose or 500 mg twice daily for 7 days.
  - **Rationale**: These are nitroimidazole antibiotics effective against anaerobic organisms, including *Trichomonas vaginalis*. They disrupt the DNA of the protozoa, leading to its death.
- **Sexual partners**: Should also be treated to prevent reinfection.
  - **Rationale**: Asymptomatic carriers can pass the infection back to the treated individual.

Procedures and Surgery

- **No surgical intervention** is typically required.
  - **Rationale**: Trichomoniasis is a treatable infection with appropriate antibiotic therapy.

Nursing Management

- **Assessment**:
  - Evaluate the patient's history, symptoms, and sexual activity.
  - **Rationale**: Identifying the risk factors and symptoms guides the management plan.
- **Intervention**:
  - Administer prescribed medications (metronidazole/tinidazole).

        - **Rationale**: Ensuring compliance with the treatment regimen helps eradicate the infection.
    - Educate the patient about completing the full course of antibiotics.
        - **Rationale**: Partial treatment can lead to resistance and recurrence.
    - Advise abstaining from sexual intercourse until treatment is complete.
        - **Rationale**: Reduces the risk of reinfection.
    - Encourage sexual partner notification and treatment.
        - **Rationale**: Helps in controlling the spread of the infection.
- **Health Teaching**:
    - Safe sex practices (use of condoms).
        - **Rationale**: Prevents future infections.
    - Importance of personal hygiene.
        - **Rationale**: Reduces the risk of pathogenic growth in the genital area.

Nursing Goals

- **Primary goal**: Eradicate the infection, prevent complications, and avoid reinfection.
    - **Rationale**: Prompt treatment can prevent further transmission and long-term complications, such as infertility.

Nursing Priorities

1. Alleviating patient discomfort (pain, itching).
    - **Rationale**: Symptomatic relief improves the patient's quality of life.
2. Preventing reinfection and the spread of the infection to partners.
    - **Rationale**: Reducing transmission is key in controlling STIs.

Nursing Actions

1. Administer antibiotics as prescribed.
    - **Rationale**: Early initiation of treatment prevents the progression of the disease.
2. Educate on sexual health and prevention strategies.
    - **Rationale**: Empowering patients with knowledge helps in

preventing reinfection and promoting health.

Prevention

- **Safe sex practices**: Consistent use of condoms. ◦ **Rationale**: Condoms act as a barrier to prevent the spread of sexually transmitted infections.
- **Limiting the number of sexual partners**.
  - **Rationale**: Reduces the exposure risk to STIs, including *Trichomonas vaginalis*.
- **Regular STI screening**: Particularly in high-risk populations. ◦ **Rationale**: Early detection helps in timely management and prevents complications.

Cervicitis: Acute and Chronic

Cervicitis is the inflammation of the cervix, the lower part of the uterus that opens into the vagina. It can occur as either an acute or chronic condition and is typically caused by infection or irritation. Acute cervicitis has a sudden onset, while chronic cervicitis develops over time due to persistent or recurrent inflammation. It is a common condition in reproductive-age women, often associated with sexually transmitted infections (STIs).

Causative Agents

1. **Infectious Causes:**
   - **Chlamydia trachomatis**: The most common bacterial cause, especially in younger women. It leads to inflammation of the cervical epithelium.
   - **Neisseria gonorrhoeae**: A frequent cause of cervicitis and can co-exist with chlamydia.
   - **Trichomonas vaginalis**: A protozoan causing frothy discharge and irritation.
     - **Herpes Simplex Virus (HSV)**: Causes recurrent inflammation, typically accompanied by genital lesions. ◦ **Human Papillomavirus (HPV)**: Often associated with chronic cervicitis and can lead to cervical dysplasia.
2. **Non-Infectious Causes:**
   - **Allergies**: Reaction to latex condoms or spermicides can irritate the cervix.
     - **Chemical Irritation**: Products like douches, soaps, or feminine hygiene products can cause inflammation.

Anatomy and Physiology

- **Cervix**: The cervix is part of the lower uterus that extends into the vagina. It functions as a barrier to infections, produces mucus, and allows passage of sperm into the uterus.
    - **Rationale**: The cervix's epithelial lining is vulnerable to pathogens during intercourse or exposure to irritants, leading to inflammation.

Pathophysiology

- **Acute Cervicitis**: Caused by direct infection or irritation, triggering an inflammatory response. The pathogens invade the cervical epithelium, causing swelling, erythema, and increased mucus production. If untreated, the infection may ascend, leading to complications like pelvic inflammatory disease (PID).
    - **Rationale**: The cervix serves as a gateway to the uterus, making it susceptible to both localized and ascending infections.
- **Chronic Cervicitis**: Occurs due to persistent infection, repeated exposure to irritants, or an untreated acute infection. It can result in long-term changes to the cervical epithelium, such as hyperplasia or metaplasia.
    - **Rationale**: Chronic inflammation may lead to epithelial changes that increase the risk of cervical dysplasia or cancer.

Incubation Period

- **Chlamydia and Gonorrhea**: Typically 1-3 weeks. •

HSV: 2-12 days.

- **Trichomoniasis**: 5-28 days.
    - **Rationale**: The incubation period varies based on the pathogen's replication cycle and immune system response before symptoms appear.

Mode of Transmission

- **Sexual Transmission**: Through vaginal, oral, or anal intercourse with an infected partner.
- **Non-Sexual**: Exposure to irritants or allergic reactions to products.
    - **Rationale**: Most causative agents are sexually transmitted pathogens, but non-infectious cervicitis can result from chemical irritants.

Risk Factors

1. **Multiple Sexual Partners**: Increases exposure to STIs. 2. **Unprotected Sex**: Increases risk of transmission of pathogens.
3. **Early Sexual Activity**: Increases risk of STIs.
4. **Douching**: Alters vaginal flora and increases the risk of infection.
5. **Immunosuppression**: Reduces the body's ability to fight infections.
    - **Rationale**: These factors disrupt the cervix's natural defenses and expose it to infectious agents.

Signs and Symptoms

1. **Vaginal Discharge**: Thick, purulent, or frothy discharge is common due to mucous production from inflamed cervical glands.
2. **Postcoital Bleeding**: The fragile, inflamed cervix bleeds easily after sexual activity.
3. **Dyspareunia**: Painful intercourse due to cervical inflammation.
4. **Dysuria**: Urinary symptoms due to irritation and proximity to the urethra.
5. **Lower Abdominal Pain**: If inflammation extends to other reproductive organs.
    - **Rationale**: The inflammation of the cervix triggers localized symptoms, and ascending infections may cause systemic symptoms.

Assessment

- **History**:
    - Sexual history, contraceptive use, and exposure to irritants are important to identify the cause of cervicitis. ◦ **Rationale**: A thorough history helps in identifying risk factors and potential sources of infection.
- **Physical Examination**:
    - **Speculum Exam**: The cervix may appear red, swollen, with purulent discharge or friable tissue.
    - **Rationale**: Visual inspection helps detect signs of inflammation and infection.

Laboratory and Diagnostic Procedures

1. **Nucleic Acid Amplification Test (NAAT)**: Detects Chlamydia and Gonorrhea.

- **Rationale**: Highly sensitive and specific for identifying bacterial STIs.

2. **Wet Mount**: Detects Trichomonas or other causes of vaginal discharge.
   - **Rationale**: Microscopic evaluation identifies organisms directly.
3. **HSV PCR Test**: Identifies herpes simplex virus.
   - **Rationale**: PCR is highly sensitive for viral DNA detection.
4. **Pap Smear**: Identifies abnormal cervical cells.
   - **Rationale**: Cervicitis can be associated with epithelial cell changes.
5. **HPV Test**: Screens for high-risk HPV strains.
   - **Rationale**: Chronic cervicitis is linked to HPV-related dysplasia.

Medical Management

1. **Antibiotics**:
   - **Azithromycin** or **Doxycycline** for Chlamydia.
   - **Ceftriaxone** for Gonorrhea.
   - **Metronidazole** for Trichomoniasis.
   - **Rationale**: Antibiotics target specific causative bacteria or protozoa.
2. **Antivirals**: Acyclovir for HSV.
   - **Rationale**: Reduces viral replication and symptoms. 3.

Avoiding Irritants: Discontinue use of offending products (e.g., douches, spermicides).

- **Rationale**: Prevents further irritation of the cervix.

Procedures and Surgery

- **Cervical Cryotherapy**: Freezing abnormal tissue in chronic cervicitis or HPV-related dysplasia.
  - **Rationale**: Removes damaged cervical tissue and promotes healing.

Nursing Management

Assessment:

- **Monitor Symptoms**: Evaluate for changes in discharge, bleeding, or pain.
  - **Rationale**: Monitoring helps identify complications or treatment effectiveness.

Interventions:

1. **Administer Medications**: Ensure adherence to prescribed

antibiotics or antivirals.
    - **Rationale**: Completes the treatment course to eradicate infection.
2. **Promote Safe Sexual Practices**: Encourage condom use and STI screening for partners.
    - **Rationale**: Reduces transmission risk and reinfection.

Health Teaching:

1. **STI Prevention**: Educate on the importance of protected sex and limiting sexual partners.
    - **Rationale**: Decreases the risk of future infections. 2.

Hygiene Practices: Avoid douching and using irritants. ◦ **Rationale**: Protects the natural vaginal flora and reduces irritation.

Nursing Goals

- **Goal**: Resolve inflammation and prevent ascending infections (e.g., PID).
    - **Rationale**: Early treatment of cervicitis prevents serious reproductive health complications.

Nursing Priority

- **Priority**: Infection control and prevention of complications. ◦ **Rationale**: Treating the infection promptly avoids its spread to other reproductive organs.

Nursing Actions

1. **Ensure Patient Adherence**: Follow up on medication compliance.
    - **Rationale**: Full adherence prevents relapse and complications.
2. **Referral to Partner Notification**: Ensure sexual partners are tested and treated.
    - **Rationale**: Prevents reinfection and broader community transmission.

Prevention

- **Safe Sexual Practices**: Regular use of condoms and routine STI screening.
    - **Rationale**: Reduces exposure to sexually transmitted pathogens.
- **Vaccination**: HPV vaccination to prevent cervical dysplasia and cancer.

- **Rationale**: Reduces the incidence of HPV-related cervicitis.

Herpes Simplex Virus (HSV)

Causative Agent:

- **Herpes Simplex Virus (HSV)** is a double-stranded DNA virus from the **Herpesviridae** family. It exists in two types:
  - **HSV-1**: Primarily causes oral lesions (cold sores) but can also cause genital infections.
  - **HSV-2**: Primarily responsible for genital herpes but can occasionally cause oral lesions.

Rationale: HSV is highly neurotropic, establishing lifelong latency in the sensory neurons after initial infection. It reactivates due to various triggers, causing recurrent episodes.

Anatomy and Physiology:

- **Skin and Mucous Membranes**: HSV infects epithelial cells in areas such as the lips, mouth, genitals, or rectum.
- **Neural Involvement**: After initial infection, HSV becomes latent in the dorsal root ganglia, particularly the trigeminal ganglia for HSV-1 and sacral ganglia for HSV-2.

Rationale: The virus travels along sensory neurons, making its way to nerve roots where it remains dormant, explaining recurrent outbreaks in the same location.

Pathophysiology:

- **Initial Infection**: HSV enters epithelial cells via skin or mucous membranes, leading to local inflammation and cellular destruction. This causes vesicular lesions.
- **Latency**: Following the initial infection, the virus migrates via peripheral nerves to sensory ganglia, where it remains dormant.
- **Reactivation**: HSV reactivates under conditions such as stress, immunosuppression, or illness, traveling back to the skin or mucous membranes, leading to recurrent lesions.

Rationale: The body's immune system controls viral replication but cannot eliminate latent virus, resulting in periodic reactivation and recurrent symptoms.

Incubation Period:

- **HSV-1**: 2 to 12 days, typically around 4 days.
- **HSV-2**: 4 to 7 days.

Rationale: The variation in incubation periods is due to the time required for viral replication and the body's immune response to manifest symptoms after initial exposure.

Mode of Transmission:

- **Direct Contact**: HSV is transmitted through direct contact with infected secretions, such as saliva (HSV-1) or genital secretions (HSV-2).
- **Asymptomatic Shedding**: HSV can be transmitted even when no lesions are present due to viral shedding. **Rationale**: The virus resides in body fluids, and asymptomatic shedding allows transmission even in the absence of active sores, making prevention challenging.

Risk Factors:

- **Sexual Contact**: Unprotected sexual activity, especially with multiple partners, increases the risk of HSV-2.
- **Immunocompromised States**: Conditions like HIV or use of immunosuppressive drugs increase the likelihood of severe or recurrent HSV infections.
- **Stress**: Physical or emotional stress can trigger HSV reactivation.

Rationale: Compromised immune systems have reduced ability to suppress viral replication, while mucosal or skin breaks facilitate viral entry.

Signs and Symptoms:

- **HSV-1**: Painful oral vesicular lesions, fever, malaise, pharyngitis, and lymphadenopathy.
- **HSV-2**: Painful genital vesicles, dysuria, and systemic symptoms such as fever and malaise.

Rationale: The immune response to viral replication causes local inflammation, while systemic symptoms reflect cytokine release.

Assessment:

- **History**: Ask about sexual history, previous occurrences of oral/genital lesions, immune status, and potential contact with infected individuals.
- **Physical Examination**: Inspect for vesicular lesions, erythema, and lymphadenopathy, especially in the oral or genital region.

Rationale: History provides clues to exposure, and physical signs

like vesicles are hallmark signs of active infection.

Laboratory and Diagnostic Procedures:

- **Polymerase Chain Reaction (PCR)**: Detects HSV DNA in lesion swabs or cerebrospinal fluid (for suspected HSV encephalitis).
- **Tzanck Smear**: Identifies multinucleated giant cells, but is less specific and sensitive than PCR.
- **Serologic Tests**: Detects antibodies to differentiate between HSV-1 and HSV-2.

Rationale: PCR is the gold standard due to its high sensitivity, while serology can help differentiate between HSV-1 and HSV-2 infection.

Medical Management:

- **Antiviral Therapy**:
  - **Acyclovir**, **Valacyclovir**, or **Famciclovir** are used to reduce symptom severity, duration, and frequency of outbreaks.

Rationale: Antivirals inhibit viral DNA synthesis, limiting viral replication and hastening healing of lesions.

Procedures and Surgery:

- **None Required**: HSV generally does not necessitate surgical intervention. However, severe HSV infections like encephalitis may require hospitalization for intravenous antiviral therapy.

Rationale: HSV infection is managed medically rather than surgically, with emphasis on controlling viral activity and managing symptoms.

Nursing Management:

1. Assessment:

- Monitor for the presence of lesions, pain, dysuria, and signs of systemic infection such as fever.

Rationale: Close monitoring allows early intervention to reduce symptom severity and prevent complications.

2. Interventions:

- Administer prescribed antiviral medications.
- Educate the patient on proper hygiene to prevent transmission.
- Apply cool compresses to soothe lesions and reduce pain.

Rationale: Antivirals limit viral replication, hygiene reduces transmission, and cool compresses can provide symptomatic relief.

3. Health Teaching:

- Advise on the use of condoms during sexual activity to reduce

transmission risk.
- Emphasize avoiding contact with lesions and frequent handwashing.
- Instruct on recognizing triggers for reactivation (stress, illness).

Rationale: Education on prevention helps reduce the spread and frequency of outbreaks, while understanding triggers helps patients manage recurrences.

Nursing Goals:

- **Goal**: To alleviate symptoms, prevent transmission, and educate the patient on the chronic nature of HSV.

Rationale: HSV is lifelong, so effective management focuses on controlling symptoms and preventing the spread to others.

Nursing Priority:

- **Pain Management and Infection Control**.

Rationale: Pain and discomfort from lesions are significant concerns, while infection control is crucial to prevent the spread of HSV to others, especially during active outbreaks.

Nursing Actions:

- **Provide Pain Relief**: Administer analgesics or apply topical anesthetics as needed.
- **Educate on Safe Practices**: Ensure patients understand how to prevent transmission, especially during asymptomatic periods.

Rationale: Pain management improves comfort, and education minimizes transmission risks to others.

Prevention:

- **Safe Sexual Practices**: Use of condoms, dental dams, and abstaining from sexual contact during outbreaks.
- **Avoiding Contact with Active Lesions**: Especially important for immunocompromised individuals and pregnant women to prevent neonatal herpes.

Rationale: HSV is easily transmissible during active outbreaks, and condoms help reduce (but not eliminate) the risk of transmission. Neonatal herpes can be life-threatening, so prevention in pregnant women is crucial.

Syphilis

Causative Agent

Treponema pallidum is the causative agent of syphilis, a spirochete bacterium. Its spiral shape and corkscrew movement allow it to burrow into mucous membranes and penetrate tissue, which makes it highly invasive. This ability to evade immune defenses is key to its pathogenicity, resulting in systemic infection.

Anatomy and Physiology

- **Infection Site**: Syphilis primarily affects the skin, mucous membranes, cardiovascular system, and central nervous system (CNS). Upon entry via skin or mucous membrane lesions, Treponema pallidum disseminates through the lymphatic system and bloodstream, impacting multiple organs.
- **Immune Response**: T. pallidum triggers both cellular and humoral immune responses. However, it evades destruction by immune cells due to its unique structure and motility, allowing it to persist in the body and cause chronic disease.

Pathophysiology

- **Primary Syphilis**: Begins with a localized infection at the site of inoculation, resulting in a painless ulcer (chancre). The bacterium multiplies locally, and the chancre appears as the body tries to limit its spread.
- **Secondary Syphilis**: After the chancre heals, T. pallidum spreads through the bloodstream, causing systemic symptoms such as skin rashes, mucous membrane lesions, and flu-like symptoms.
- **Latent Syphilis**: The bacterium may become dormant without treatment, leading to an asymptomatic phase, but it remains in the body.
- **Tertiary Syphilis**: If untreated, this late stage involves severe complications affecting the heart, CNS, and other organs, leading to gummas (granulomatous lesions), neurosyphilis, and cardiovascular syphilis.

Incubation Period

- **10-90 days (average of 21 days)**. The long incubation period allows the bacteria to proliferate at the site of entry before the immune system reacts, which is why the chancre appears after a delay.

Mode of Transmission

- **Sexual contact** (vaginal, anal, or oral), **transplacental transmission** from mother to fetus (congenital syphilis), and **contact with infected lesions** are the primary modes of transmission. Treponema pallidum cannot survive outside the human body for long, so direct mucosal or skin contact is required for transmission.

Risk Factors

- **Unprotected sexual activity**: Increases the risk of exposure to infected lesions.
- **Multiple sexual partners**: Heightens the chance of encountering an infected individual.
- **Men who have sex with men (MSM)**: Have a higher prevalence due to sexual practices and contact patterns.
- **Previous history of sexually transmitted infections (STIs)**: Indicates high-risk sexual behavior.
- **Pregnancy**: Untreated syphilis in pregnancy can result in congenital syphilis.

Signs and Symptoms

- **Primary Syphilis**: Painless chancre at the infection site, typically the genitals, anus, or mouth. The absence of pain may delay diagnosis.
- **Secondary Syphilis**: Systemic symptoms including a non itchy rash (often on the palms and soles), mucous patches in the mouth, flu-like symptoms (fever, sore throat), and lymphadenopathy. These symptoms reflect the spread of the bacteria throughout the body.
- **Tertiary Syphilis**: Symptoms include gummas (destructive lesions), neurosyphilis (dementia, ataxia, paralysis), and cardiovascular complications like aortic aneurysms. This stage reflects severe damage to tissues and organs due to chronic inflammation.

Assessment

- **History**: Assess for high-risk behaviors (unprotected sex, multiple partners, MSM), past STIs, and symptoms like skin rashes or ulcers.
- **Physical Examination**: Inspect for a painless chancre (primary syphilis), rashes, mucous patches, lymphadenopathy, and neurological signs like altered mental status or ataxia in late

syphilis.

Laboratory and Diagnostic Procedures

- **Nontreponemal Tests**: RPR (Rapid Plasma Reagin) and VDRL (Venereal Disease Research Laboratory) tests are used for screening, as they detect antibodies against cardiolipin, a substance released by damaged cells.
- **Treponemal Tests**: FTA-ABS (Fluorescent Treponemal Antibody Absorption) and TPPA (Treponema pallidum Particle Agglutination) confirm syphilis diagnosis by detecting specific antibodies against T. pallidum.
- **Lumbar Puncture**: Performed if neurosyphilis is suspected, to analyze cerebrospinal fluid (CSF) for T. pallidum.

Medical Management

- **Penicillin G (Benzathine penicillin)**: The antibiotic of choice, it directly kills T. pallidum by inhibiting cell wall synthesis. For neurosyphilis, higher doses of penicillin are required to penetrate the CNS.
- **Alternative Antibiotics**: For penicillin-allergic patients, doxycycline or tetracycline may be used, although penicillin desensitization is preferred due to the efficacy of penicillin in treating syphilis.

Procedure and Surgery

- **No routine surgical interventions** are required for syphilis treatment, except in the late stage (tertiary syphilis) when surgical repair of damage (e.g., aneurysms, gummas) may be necessary. Neurosyphilis may require specialized care if neurological damage is severe.

Nursing Management

- **Assessment**: Ongoing assessment of treatment effectiveness (resolution of symptoms) and monitoring for potential Jarisch-Herxheimer reaction (an acute febrile reaction occurring after antibiotic treatment due to bacterial endotoxin release).
- **Intervention**: Administer prescribed antibiotics, provide education on medication adherence, and emphasize the need for sexual partner notification and treatment to prevent reinfection.
- **Health Teaching**: Educate patients on safe sexual practices (condom use, limiting sexual partners), importance of follow up

testing, and the potential for serious complications if untreated.

Nursing Goal

- The primary goal is **to eradicate the infection and prevent its transmission**. Early detection and treatment minimize complications and stop disease progression.

Nursing Priority

- **Immediate antibiotic administration** is the priority to halt disease progression and reduce transmission. Early-stage syphilis is highly treatable, but delayed treatment increases the risk of severe, irreversible complications.

Nursing Action

- **Initiate treatment as prescribed**, monitor for adverse reactions (e.g., Jarisch-Herxheimer reaction), and support the patient in contacting partners for evaluation and treatment to prevent reinfection and further spread of the disease.

Prevention

- **Safe sexual practices**: Consistent condom use reduces exposure to syphilis sores.
- **Regular STI screening**: High-risk individuals should be screened regularly, allowing for early detection and treatment. •
Partner notification: Informing sexual partners to get tested and treated prevents reinfection and curtails the disease's spread.

Gonorrhea

Causative Agent

Gonorrhea is caused by *Neisseria gonorrhoeae*, a gram-negative diplococcus bacterium. *N. gonorrhoeae* attaches to the mucosal surfaces of the genitourinary tract, eyes, throat, or rectum via pili and induces an inflammatory response. The bacterium multiplies in these areas, causing purulent discharge. Its pathogenicity arises from the ability to evade host immunity and cause tissue damage by releasing endotoxins.

Anatomy and Physiology

Gonorrhea primarily affects the mucous membranes of the genitourinary tract, including the urethra in men and the endocervix in women. It can also infect the rectum, oropharynx, and conjunctiva.

The bacteria invade these epithelial cells, leading to an immune response characterized by inflammation and pus formation. In women, the infection can spread to the fallopian tubes and ovaries, causing pelvic inflammatory disease (PID), leading to infertility or ectopic pregnancy if left untreated.

Pathophysiology

Once *N. gonorrhoeae* enters the body, it attaches to and penetrates mucosal epithelial cells. This invasion triggers an immune response that leads to neutrophil influx, resulting in inflammation and purulent discharge. The infection can ascend the reproductive tract, potentially causing PID in women, and may disseminate, leading to conditions such as septic arthritis and systemic gonococcal infections if not treated. The body's immune response is often insufficient to clear the infection without medical intervention due to the bacteria's ability to avoid immune detection.

Incubation Period

The incubation period for gonorrhea ranges from 2 to 14 days, with most individuals developing symptoms within 2 to 5 days after exposure. This time frame is related to how long it takes for the bacteria to replicate sufficiently to cause noticeable symptoms.

Mode of Transmission

Gonorrhea is transmitted primarily through sexual contact, including vaginal, oral, and anal sex. The bacterium can also be passed from mother to child during childbirth, causing neonatal conjunctivitis. Transmission occurs when *N. gonorrhoeae* comes into contact with the mucous membranes of a new host. This direct contact is essential for infection as the bacterium cannot survive long outside the body.

Risk Factors

- Multiple sexual partners: Increased exposure to infected individuals raises the risk of contracting gonorrhea.
- Unprotected sex: Lack of condom use facilitates the spread of *N. gonorrhoeae*.
- Adolescence: Younger individuals, particularly women aged 15-24, are more biologically susceptible to infection.
- Previous sexually transmitted infections (STIs): Having a history of STIs can make individuals more susceptible due to damaged mucosal surfaces.

Signs and Symptoms

- **Men**: Urethritis, purulent discharge from the penis, and dysuria are common. These occur due to inflammation and pus formation at the infection site.
- **Women**: Symptoms include vaginal discharge, dysuria, and intermenstrual bleeding. These arise from infection and inflammation of the cervix and urethra. Many women are asymptomatic, which increases the risk of complications like PID.
- **Rectal Infection**: Symptoms include anal itching, rectal discharge, and discomfort, all resulting from local inflammation.
- **Oropharyngeal Infection**: It may cause a sore throat, difficulty swallowing, and pus in the tonsillar area.

Assessment: History and Physical Examination

- **History**: Obtain a detailed sexual history, including the number of sexual partners, use of protection, and prior history of STIs. This helps identify exposure risks.
- **Physical Examination**: In men, inspect for urethral discharge and perform a genital examination. In women, a pelvic examination should be conducted to assess for signs of cervicitis or PID. Examination of the throat, rectum, and eyes is also critical if symptoms suggest extra-genital involvement.

Laboratory and Diagnostic Procedures

- **Nucleic Acid Amplification Test (NAAT)**: This is the gold standard for diagnosing gonorrhea due to its high sensitivity and specificity. It can be used on urine samples or swabs from the cervix, urethra, rectum, or throat.
- **Gram Stain**: In symptomatic men, a Gram stain of urethral discharge may reveal intracellular gram-negative diplococci, confirming the diagnosis.
- **Culture**: In cases of treatment failure or antibiotic resistance, culturing *N. gonorrhoeae* helps guide therapy.

Medical Management

- **Antibiotics**: Current guidelines recommend dual therapy with ceftriaxone (250 mg intramuscularly) and azithromycin (1 g orally). Ceftriaxone targets the bacterium's cell wall, while azithromycin covers potential coinfection with *Chlamydia trachomatis*. Treatment must be prompt to prevent

complications like PID or disseminated gonococcal infection.

Procedure and Surgery

No surgical interventions are typically required for gonorrhea unless complications like tubo-ovarian abscesses develop, which may necessitate drainage. In cases of neonatal conjunctivitis, ophthalmic ointments containing antibiotics are applied to prevent blindness.

Nursing Management

- **Assessment**: Monitor the patient's symptoms, especially discharge, dysuria, and pelvic pain. Early identification of complications like PID or disseminated gonococcal infection is crucial.
- **Intervention**: Administer antibiotics as prescribed, ensuring the patient understands the importance of completing the course of treatment. Provide pain relief for symptomatic relief.
- **Health Teaching**: Educate patients on the importance of partner notification and treatment to prevent reinfection and further transmission. Encourage safe sexual practices, including condom use. Advise patients to abstain from sexual activity until the infection is fully treated.

Nursing Goal

The primary goal is to eradicate the infection and prevent complications such as PID or systemic gonococcal infection. This is achieved by ensuring adherence to prescribed antibiotics, monitoring for adverse effects, and promoting safe sexual practices.

Nursing Priority

The immediate priority is preventing the spread of infection and addressing any complications. Rapid initiation of antibiotic therapy and education on prevention strategies (e.g., condom use and partner notification) are crucial to reducing transmission and morbidity.

Nursing Actions

- Collect appropriate specimens for testing to confirm the diagnosis.
- Administer antibiotics promptly to minimize complications. • Educate the patient on adherence to the treatment regimen and

the need for follow-up to ensure the infection is eradicated.
- Provide emotional support, as an STI diagnosis can carry social and emotional stigma.

Prevention

- **Safe Sexual Practices**: Consistent condom use reduces the risk of transmission.
- **Routine Screening**: Regular screening for sexually active individuals, especially those with multiple partners, is essential for early detection and treatment.
  - **Partner Notification**: Prompt treatment of sexual partners prevents reinfection and halts the spread of the disease.

Human Papillomavirus (HPV)

Causative Agent

HPV is caused by the **human papillomavirus**, a DNA virus from the Papillomaviridae family. There are over 200 types of HPV, with HPV-16 and HPV-18 being the most oncogenic types, primarily associated with cervical cancer. Other types (e.g., HPV-6 and HPV-11) are commonly linked to benign genital warts. The virus infects epithelial cells and promotes abnormal cellular proliferation, leading to warts, dysplasia, and malignancy depending on the strain.

Anatomy and Physiology

HPV primarily affects the **epithelial tissues** of the skin and mucous membranes, including the genital, anal, oropharyngeal, and respiratory tracts. It targets the **basal cells** of stratified squamous epithelium. In the cervix, HPV can infect the **transformation zone**, where columnar epithelium transitions to squamous epithelium, an area particularly prone to malignant transformation. Infections occur when microtraumas expose basal cells, allowing viral entry and replication.

Pathophysiology

HPV enters the body through small abrasions in the skin or mucosa and infects basal epithelial cells. Once inside the cell, the virus hijacks the host's cellular machinery to replicate. Low-risk HPV types (e.g., HPV-6, HPV-11) cause benign proliferative lesions like genital warts. High-risk HPV types (e.g., HPV-16, HPV-18) integrate their DNA into the host genome, disrupting cell cycle regulation by inactivating tumor

suppressor proteins p53 and Rb, which leads to uncontrolled cell division, DNA damage, and oncogenesis (e.g., cervical, anal, and oropharyngeal cancers).

Incubation Period

The incubation period for HPV can vary but typically ranges from **2 weeks to 8 months** after exposure. This prolonged incubation period reflects the virus's slow replication and the latency before symptomatic manifestations like warts or dysplastic lesions. In some cases, the virus remains dormant for years without causing symptoms.

Mode of Transmission

HPV is primarily transmitted through **direct skin-to-skin contact**, most commonly through vaginal, anal, or oral sex. Non-sexual transmission is possible, such as via contaminated surfaces or vertical transmission from mother to child during birth. The virus's ability to infect basal epithelial cells through microscopic abrasions makes it highly contagious, even in the absence of visible lesions.

Risk Factors

- **Multiple sexual partners**: Increases exposure risk to different HPV strains.
- **Early sexual activity**: Exposure to HPV at a younger age correlates with a longer duration of infection, increasing cancer risk.
- **Immunocompromised status**: Conditions like HIV reduce the body's ability to clear HPV, leading to persistent infection.
- **Unprotected sex**: Barrier methods, though not fully protective, reduce HPV transmission.
- **Smoking**: Impairs immune response, increasing the persistence of HPV infection.
- **Co-infection with other STIs**: Heightens susceptibility to HPV and complicates immune response.

Signs and Symptoms

- **Genital warts**: Painless, fleshy growths on genital or anal areas due to HPV-6 and HPV-11.
- **Cervical dysplasia**: Often asymptomatic, detected through screening.
- **Oropharyngeal lesions**: Sore throat or voice changes due to

HPV-16.

- **Cancer symptoms**: Abnormal vaginal bleeding, pelvic pain, or discharge in cervical cancer; dysphagia and throat pain in oropharyngeal cancer.

Assessment

- **History**: Sexual activity, use of protection, number of sexual partners, smoking history, prior STIs, and immunization status (HPV vaccine).
- **Physical Examination**: Visual inspection for warts, abnormal lesions on genitalia, oropharyngeal, or anal areas. Pap smears to identify cervical dysplasia.

Laboratory and Diagnostic Procedures

- **Pap smear**: Detects cervical dysplasia (precancerous changes) in the cervix.
- **HPV DNA test**: Identifies high-risk HPV strains linked to cancer.
- **Colposcopy**: Magnified examination of the cervix to identify suspicious lesions.
- **Biopsy**: Confirms malignancy in abnormal lesions. • **PCR testing**: Molecular technique to identify specific HPV strains.

Medical Management

- **Topical agents**: Imiquimod or podofilox for genital warts. • **Cryotherapy or electrocautery**: To remove warts. • **Antiviral treatment**: Currently, no antiviral treatment exists to cure HPV, but vaccination (e.g., Gardasil, Cervarix) prevents infection from common high-risk and low-risk strains. • **Cancer treatment**: Chemotherapy, radiation, or surgery in cases of HPV-related cancers.

Procedure and Surgery

- **LEEP (Loop Electrosurgical Excision Procedure)**: Removes abnormal cervical tissue before progression to cancer.
- **Laser therapy**: Used to ablate warts or precancerous lesions. • **Conization**: A surgical procedure to remove a cone-shaped portion of the cervix with precancerous cells.

Nursing Management

Assessment:

- **History**: Collect a detailed sexual history, vaccination status,

and any symptoms related to HPV infection.

- **Physical Examination**: Inspect genital, oral, and anal areas for lesions or warts.

Intervention:

- **Wart treatment**: Provide care post-cryotherapy, laser, or LEEP procedures, monitoring for complications like bleeding or infection.
- **Emotional support**: Many patients feel anxiety and stigma around HPV infection, so providing reassurance is crucial. • **HPV vaccination**: Educate patients about the benefits of HPV vaccination for prevention, especially in adolescents before sexual debut.

Health Teaching:

- **Safe sexual practices**: Use of condoms to reduce transmission risk.
- **HPV vaccination**: Encourage vaccination for boys and girls aged 11–12 and catch-up vaccines up to age 26.
- **Screening**: Regular Pap smears for early detection of cervical dysplasia.

Nursing Goals

- **Prevent progression of HPV-related lesions**: Through early detection and treatment, the goal is to prevent benign lesions from advancing to cancer.
- **Promote safe sexual practices**: To reduce HPV transmission.
- **Educate about prevention**: Vaccination, regular screenings, and awareness of risk factors.

Nursing Priority

- The priority is **early detection and prevention of complications** such as cervical cancer by promoting regular screenings and encouraging HPV vaccination. Additionally, addressing the psychological impact and stigma associated with HPV is important.

Nursing Action

- **Administer HPV vaccine**: Direct action to prevent infection with high-risk and low-risk HPV types.
- **Educate on signs of complications**: Teaching patients to recognize symptoms like abnormal bleeding or warts can facilitate early medical intervention.

Prevention

- **HPV vaccination**: The most effective primary prevention strategy, significantly reducing the risk of genital warts and cervical cancer.
- **Barrier contraception**: Though not 100% protective, it reduces exposure to the virus.
- **Regular screening**: Pap smears and HPV DNA tests allow early identification of dysplasia, which can prevent cancer.

Human immunodeficiency virus (HIV)

Infection is caused by HIV, which is a retrovirus that causes immunosuppression. The viral infection causes the person to be susceptible to infections that would normally be controlled through immune responses. The term HIV disease is used interchangeably with HIV infection. With advances in treatment, HIV is managed as a chronic disease, since people are living longer.

MODE OF TRANSMISSION

through contact with infected blood or blood products during transfusion or tissue transplantation or by sharing a contaminated needle

through contact with infected body fluids, such as semen and vaginal fluids, during unprotected sex

Across the placental barrier from an infected mother to a fetus, or from an infected mother to an infant either through cervical or blood contact at delivery or through breast milk.

STAGES

Primary infection (Acute/Recent HIV Infection). The period from infection with HIV to the development of HIV- specific antibodies is known as primary infection.

HIV asymptomatic (CDC Category A). After the viral set point is reached, HIV-positive people enter into a chronic stage in which the immune system cannot eliminate the virus despite its best efforts.

HIV symptomatic (CDC Category B). Category B consists of symptomatic conditions in HIV-infected patients that are not included in the conditions listed in category C.

AIDS (CDC Category C). When the CD4+ T-cell level drops

below 200 cells/mm3 of blood, the person is said to have AIDS.

PATHOPHYSIOLOGY

In this first step, the GP120 and GP41 glycoproteins of HIV bind with the host's uninfected CD4+ receptor and chemokine coreceptors, usually CCR5, which results in fusion of HIV with the CD4+ T-cell membrane.

The contents of HIV's viral core are emptied into the CD4+ T cell. DNA synthesis. HIV changes in genetic material from RNA to DNA through action of reverse transcriptase, resulting in double stranded DNA that carries instruction for viral replication. New viral DNA enters the nucleus of the CD4+ T cell and through action of integrase is blended with the DNA of the CD4+ T cell, resulting in permanent, lifelong infection.

When the CD4+ T cell is activated, the double-stranded DNA forms single-stranded messenger RNA, which builds new viruses. The mRNA creates chains of new proteins and enzymes that contain the components needed in the construction of new viruses. The HIV enzyme protease cuts the polyprotein chain into the individual proteins that make up the new virus.

New proteins and viral RNA migrate to the membrane of the infected CD4+ T cell, exits from the cell, and starts the process all over.

COMPLICATIONS

Opportunistic infections. Patients who are immunosuppressed are at risk for opportunistic infections such as pneumocystis pneumonia which can affect 80% of all people infected with HIV.

Respiratory failure. Impaired breathing is a major complication that increases the patient's discomfort and anxiety and may lead to respiratory and cardiac failure.

Cachexia and wasting. Wasting syndrome occurs when there is profound involuntary weight loss exceeding 10% of the baseline body weight and it is a common complication of HIV infection and AIDS.

Viral Hepatitis A, B, C, D, and E

Viral hepatitis is an inflammation of the liver caused by different viruses: hepatitis A (HAV), hepatitis B (HBV), hepatitis C (HCV), hepatitis D (HDV), and hepatitis E (HEV). Each virus affects the liver differently and has unique transmission routes, pathophysiology, clinical presentations, and management approaches.

Causative Agent

- **Hepatitis A**: HAV, a non-enveloped RNA virus, primarily transmitted via the fecal-oral route.
- **Hepatitis B**: HBV, a DNA virus, can cause both acute and chronic hepatitis and is transmitted via blood and bodily fluids.
- **Hepatitis C**: HCV, an RNA virus, often leads to chronic infection and is primarily bloodborne.
- **Hepatitis D**: HDV, an incomplete RNA virus, requires HBV co-infection to replicate.
- **Hepatitis E**: HEV, a non-enveloped RNA virus, is similar to HAV in its transmission route, primarily fecal-oral.

Rationale: These viruses are hepatotropic, meaning they specifically target liver cells, causing inflammation and damage.

Anatomy and Physiology

The liver is vital for metabolism, detoxification, bile production, and storage of nutrients. Hepatitis viruses cause liver inflammation, leading to impaired liver function.

- **Liver structure**: Comprised of lobes, hepatocytes, bile ducts, and blood vessels.
- **Functions**: Synthesizes clotting factors, processes toxins, stores glycogen, and metabolizes lipids, proteins, and carbohydrates.

Rationale: Understanding the liver's role helps comprehend how viral infections disrupt normal function, causing systemic effects like jaundice and coagulopathy.

Pathophysiology

- **Hepatitis A**: Viral entry into hepatocytes leads to immune mediated hepatocellular damage. There is no chronic phase.
- **Hepatitis B**: Can lead to acute or chronic inflammation. In chronic cases, ongoing immune responses cause fibrosis and cirrhosis.
- **Hepatitis C**: Chronic inflammation from HCV leads to progressive liver damage, fibrosis, and cirrhosis.
- **Hepatitis D**: Co-infection with HBV accelerates liver damage, increasing the risk for cirrhosis.
- **Hepatitis E**: Similar to HAV, causes acute liver inflammation without chronic sequelae in most cases.

Rationale: The liver damage seen in viral hepatitis results from the immune response to infected hepatocytes rather than the virus itself directly killing liver cells.

Incubation Period

- **Hepatitis A**: 15–50 days (average 28 days).
- **Hepatitis B**: 45–180 days (average 60–90 days).
- **Hepatitis C**: 14–180 days (average 45 days).
- **Hepatitis D**: 30–180 days.
- **Hepatitis E**: 15–60 days (average 40 days).

Rationale: Knowledge of the incubation period is essential for tracking potential exposures and initiating appropriate preventive measures.

Mode of Transmission

- **Hepatitis A and E**: Fecal-oral route, often through contaminated water or food.

- **Hepatitis B, C, and D**: Bloodborne; transmitted through blood, sexual contact, and perinatally.

Rationale: Different modes of transmission require distinct preventive strategies, such as vaccination for HAV and HBV, and safe injection practices for HCV and HDV.

Risk Factors

- **Hepatitis A**: Poor sanitation, traveling to endemic areas, contaminated food.
- **Hepatitis B**: Unprotected sex, sharing needles, healthcare exposure, mother-to-child transmission.
- **Hepatitis C**: Injection drug use, blood transfusions before 1992, organ transplants, unsterilized medical equipment. • **Hepatitis D**: Co-infection with HBV, drug use.
- **Hepatitis E**: Poor sanitation, drinking contaminated water.

Rationale: Recognizing risk factors enables early identification of at-risk populations for screening and preventive interventions.

Signs and Symptoms

- **Acute**: Fatigue, nausea, vomiting, abdominal pain, jaundice, dark urine, clay-colored stools, joint pain, and fever. • **Chronic (HBV, HCV, HDV)**: Often asymptomatic until liver damage is severe, presenting with jaundice, ascites, spider angiomas, and hepatic encephalopathy.

Rationale: Symptoms are due to impaired liver function, particularly the accumulation of bilirubin (jaundice), decreased bile production, and immune response to infection.

Assessment: History and Physical Examination • **History**: Inquire about exposure risks (travel, sexual activity, drug use, blood transfusions), vaccination status, and family history.

- **Physical Exam**: Look for jaundice, hepatomegaly, right upper quadrant tenderness, ascites, and signs of chronic liver disease (spider angiomas, palmar erythema).

Rationale: A thorough history and physical help identify the cause, extent of liver involvement, and possible complications.

Laboratory and Diagnostic Procedures

- **Serological tests**: Specific antibody and antigen testing for each virus (e.g., HBsAg for HBV, anti-HCV for HCV). • **Liver function tests (LFTs)**: ALT, AST, bilirubin, and ALP levels are elevated in hepatitis.

- **Viral load testing**: HBV DNA, HCV RNA quantify viral replication.
- **Imaging**: Ultrasound, CT, or MRI for assessing liver damage.

Rationale: These tests confirm the diagnosis, determine the severity of infection, and guide treatment decisions.

Medical Management

- **Hepatitis A and E**: Supportive care (hydration, rest, nutritional support), as these are self-limiting.
- **Hepatitis B and C**: Antiviral therapy (e.g., entecavir, tenofovir for HBV; direct-acting antivirals for HCV).
- **Hepatitis D**: Treatment of HBV with antivirals (pegylated interferon may be used).

Rationale: Managing chronic hepatitis reduces viral load, prevents liver damage, and improves long-term outcomes.

Procedures and Surgeries

- **Liver Biopsy**: Sometimes performed to assess the extent of liver damage.
- **Liver Transplantation**: Considered in cases of end-stage liver disease or fulminant hepatitis (particularly with HBV, HCV, or HDV).

Rationale: Procedures like biopsies confirm the extent of liver damage, and transplantation is life-saving in severe liver failure.

Nursing Management

- **Assessment**: Monitor liver function tests, signs of jaundice, changes in mental status (encephalopathy), and fluid balance (ascites).
- **Interventions**: Provide rest, encourage fluid intake, monitor medication side effects (antivirals), and educate about transmission prevention and lifestyle modifications (avoiding alcohol, safe sexual practices).
- **Health Teaching**: Educate about vaccination (HAV, HBV), safe needle use, and avoiding contaminated food/water. **Rationale**: Early intervention prevents complications, while health education empowers patients to manage their condition and prevent transmission.

Nursing Goals

- Prevent disease progression.

- Promote patient education on disease transmission and lifestyle modifications.
- Ensure effective symptom management and treatment adherence.

Rationale: Proper nursing goals focus on mitigating liver damage, reducing transmission risk, and optimizing patient outcomes.

Nursing Priority

- Immediate priority is symptom relief and prevention of complications (e.g., encephalopathy, ascites).
- Long-term priority is patient education to prevent further liver damage and transmission.

Rationale: Prioritizing these aspects prevents acute decompensation and reduces the chronic burden of the disease.

Nursing Actions

- Administer antiviral medications as prescribed.
- Encourage vaccination where applicable.
- Monitor liver function and fluid-electrolyte balance closely. • Educate on hand hygiene (HAV, HEV) and safe practices (HBV, HCV, HDV).

Rationale: These actions are vital in managing symptoms, preventing complications, and educating the patient about long term health maintenance.

Prevention

- **Hepatitis A and E**: Safe drinking water, proper sanitation, vaccination (HAV).
- **Hepatitis B**: Vaccination, safe sex practices, avoiding needle sharing, screening blood donors.
- **Hepatitis C**: No vaccine available; focus on bloodborne precautions.
- **Hepatitis D**: HBV vaccination prevents HDV infection. • **Hepatitis E**: Improved sanitation and avoiding contaminated water sources.

Rationale: Prevention strategies reduce the incidence of viral hepatitis, especially through vaccination and behavior modification.