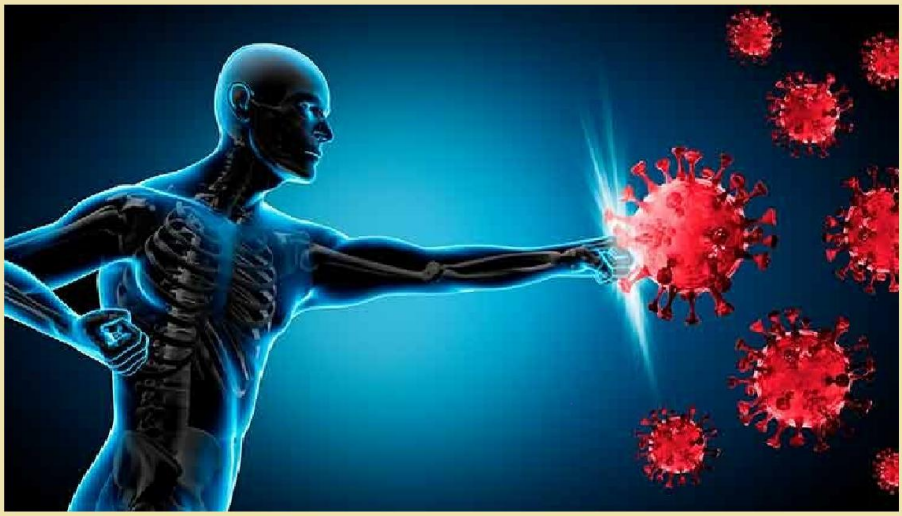
April-2024

S.TENTISHEV ASIAN MEDICAL INSTITUTE

Kant-Kyrgyzstan

Inter-Professional Discipline Department



T.S.A.M

"THE STUDENT'S ABSTRACT OF MEDICINE"

FOR

Interpreofessional Communication And Partnership In Health Care & Medical Education

4th

Dr.AFTAB SHEIKH
Senior Lecturer
Orthopedic Surgeon & Traumatologist

AsMI Pharmacological Society

S. Tenishev Asian Medical Institute

ABSTRACT

Department of Interprofessional Disciplines

A collection of works by AzMI students under the guidance of

Senior lecturer Dr.Aftab Sheikh in the specialty

General Medicine & Dentistry

1-Collection of works by AzMI students under the guidance of Senior lecturer ***Dr.Aftab Sheikh*** was approved and recommended in meeting of
The Department of Interprofessional Disciplines ,Protocol No.01 ,dated 5th of September 2023 academic year.
2-***Approved by Head of the Department Ryspekova Altynay Erkinbekovna on specialities GENERAL MEDICINE 560001 and Stamotology 540004***
3-The collection consists of 40-45 pages.
4-The collection will be published 7 times in an academic year
5-Release date from the 20th to 30th of the month.
6-Work of students of 3-4-5 years of 5 and 6 year programs as included and recommended
7-Relevance of the collection of information about the importance of thetopic of disease of organ systems and other diseases like infections etc.
8-Rules and requirement for students research paper students submit their topic by the 10th of the month included REFERENCES

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MOHAMMAD SAMEER
3RD Year 5th Semester (2021-2026)

Neurosyphilis

Neurosyphilis is a manifestation of syphilis infection caused by the bacterium Treponema pallidum, involving the central nervous system. It can occur at any stage of syphilis, but most commonly presents in the tertiary stage of the disease.

PATHOPHYSIOLOGY:

- The bacteria invade the central nervous system, potentially causing meningitis, vasculitis, or parenchymal damage.
- Neurological involvement can lead to a wide range of symptoms, depending on the specific areas of the brain or spinal cord affected.

TRANSMISSION:

Syphilis is transmitted during oral, anal or vaginal sex through contact with infectious lesions, and also during pregnancy through the placenta. Transmission typically occurs during early stages of the disease, i.e., up to 2 years after infection.

CLINICAL FORMS:

- Asymptomatic neurosyphilis: No symptoms are evident, but cerebrospinal fluid (CSF) abnormalities are present.
- Meningovascular neurosyphilis: Inflammation of the meninges and blood vessels can lead to ischemic events such as stroke.
- General paresis: Progressive inflammation and degeneration of cerebral cortex, leading to dementia and motor dysfunction.
- Tabes dorsalis: Degeneration of the dorsal columns and dorsal roots of the spinal cord, resulting in ataxia and loss of proprioception.

RISK FACTORS:

Common risk factors in the development of neurosyphilis include:

- HIV infection
- Male gender
- High serum rapid plasma reagent titer

Less common risk factors in the development of neurosyphilis include:

- Advanced age
- African American race

DIAGNOSIS:

- Diagnosis involves serological tests for syphilis, such as the rapid plasma reagin (RPR) or Venereal Disease Research Laboratory (VDRL) test, confirmed by treponemal-specific tests.
- Lumbar puncture is performed to analyze CSF for pleocytosis, elevated protein, and a positive VDRL test, which is highly specific for neurosyphilis.

TREATMENT:

- Penicillin G administered intravenously is the treatment of choice, often requiring hospitalization for adequate dosing and monitoring.
- Follow-up includes clinical evaluation and CSF analysis to ensure therapeutic response.

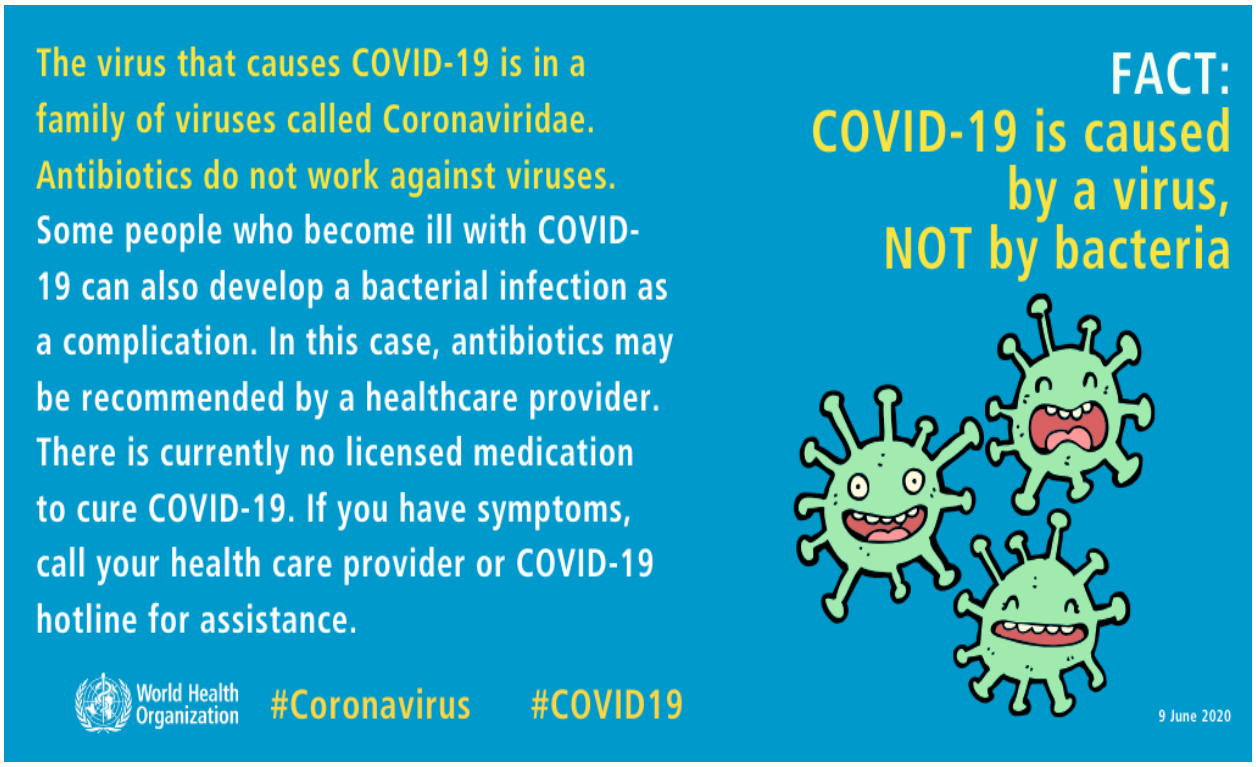
Early detection and treatment of syphilis are critical to prevent neurosyphilis. Even with treatment, some neurological damage may be irreversible, highlighting the important of prevention and early intervention.

REFERENCE:

1-https://www.who.int/news-room/fact-sheets/detail/syphilis

2-Textbook of Microbiology and Immunology 2nd Edition Subhash Chandra Parija (ELSEVIER)

3- https://www.wikidoc.org/index.php/Neurosyphilis



GAURAV KUMAR
3rd Year 6th Semester(2022-2026) MBBS

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Trichomonas Vaginalis

Trichomonas vaginalis is estimated to be the most common non-viral sexually transmitted infection (STI) among women worldwide. Infection in men is traditionally thought to be benign and self-limited.

EPIDEMOLOGY

In 2016, the World Health Organization estimated the global prevalence of T. vaginalis to be 5.0% in women and 0.6% in men. These data demonstrate a geographically variable burden of trichomoniasis among men, with prevalence ranging from 0.2% to 1.3%, with the highest rates occurring in Africa and the Americas . In the United States, T. vaginalis incidence among men was estimated to be 4.1 million infections in 2018 . Data from the 2013–2014 cycle of the National Health and Nutrition Examination Survey (NHANES) estimated the US T. vaginalis prevalence in men ages 18–59 years to be 0.5% compared with 1.8% among women . This study was the first to provide national prevalence data for T. vaginalis in US men. In addition, it was the first time NHANES utilized the highly sensitive and specific Hologic Gen-Probe Aptima T. vaginalis nucleic acid amplification test (NAAT) on urine specimens for diagnosis in both women and men . Given the sexually transmitted nature of T. vaginalis, the prevalence in men and women should be approximately equivalent.

PATHOGENICITY

The *T vaginalis* parasite is a single-celled protozoan with 4 flagella at one end. Under a microscope, these flagella may be seen propelling the parasite. Infection may produce local inflammation as parasites adhere to mucosal tissue. *T vaginalis* parasites can infect both women and men, and are passed readily between sex partners, usually during penile-vaginal sex.

Clinical signs and symptoms of trichomoniasis are variable and may include

- Itching or irritation
- Local erythema
- Burning sensation during urination or ejaculation
- Vaginal or urethral frothy discharge that may be any color but is classically yellow/green and malodorous
- None, because 70% to 85% of infected persons are asymptomatic

Initial symptoms may develop within 5 to 28 days. However, untreated infections can last for months to years, and symptoms might occur at any time.

DIAGNOSIS

Several newly available diagnostic assays may improve the ability to identify *T vaginalis* infections in comparison with traditional methods. Available diagnostic methods include the following:

- APTIMA Trichomonas vaginalis assay, a highly sensitive nucleic acid amplification test (NAAT)
- OSOM Trichomonas Rapid Test, a dipstick that can be used at the point of care
- Affirm VPIII, a nucleic acid probe that assesses 3 microbial causes of vaginitis
- Wet mount microscopy, a common low-cost test with poor sensitivity
- Culture, the traditional gold-standard method

Depending on the type of esophagitis you have, you may lessen symptoms or stop recurring problems by following these steps:

1. Do not eat foods that may increase reflux. ...
2. Use good pill-taking habits. ...
3. Lose weight. ...
4. If you smoke, quit. ...
5. Try not to stoop or bend, especially soon after eating.
6. Do not lie down after eating.

TREATMENT

- Recommended Regimens
- Metronidazole: 2 g orally in a single dose
- OR
- Tinidazole: 2 g orally in a single dose
- Alternative Regimen
- Metronidazole: 500 mg orally twice a day for 7 days.

PREVENTION

Approaches to preventing trichomoniasis include:

- Abstaining from sex
- Using condoms
- Ensuring that all sex partners receive adequate treatment
- Refraining from douching

STDs, including trichomoniasis, can be avoided by abstaining entirely from sex. Among sexually active individuals, however, a more realistic approach may be to use condoms consistently and correctly.

All sex partners of a person diagnosed with *T vaginalis* infection should be notified promptly and treated appropriately before resuming sexual activity. Patient-delivered partner therapy has been found to be as effective as standard notification, and is an option in states where this strategy is permissible

CONCLUSION

Although *T vaginalis* infection is quite common, and usually curable with a widely available and fairly inexpensive medication, a lack of public awareness makes trichomoniasis a “neglected” STD. Disparities in the prevalence of infection by sex, age, race/ethnicity, and setting should be recognized. The emergence of antimicrobial resistance and lack of alternative treatments is of concern. Additional data regarding the severity and costs of infection, as well as evidence that treatment of *T vaginalis* can prevent associated conditions, could lead to wider recognition of this infection in the future

KEY POINTS

- Although *Trichomonas vaginalis* is the most prevalent curable sexually transmitted infection, it has been considered a “neglected” parasitic infection, due to limited knowledge of its sequelae and associated costs.
- Newly available diagnostic methods, including nucleic acid amplification tests, may improve the ability to identify trichomoniasis in the clinical setting.
- Infections usually can be cured with a single oral dose of a nitroimidazole antimicrobial (eg, metronidazole or tinidazole). Allergy and antimicrobial resistance are of concern, given the lack of effective treatment alternatives.
- Prevention approaches include condoms and treatment for all sex partners.

REFERENCE

Swygard H, Seña AC, Hobbs MM, Cohen MS (April 2004). "Trichomoniasis: clinical manifestations, diagnosis and management". Sex Transm Infect. 80 (2): 91–5. doi:10.1136/sti.2003.005124. PMC 1744792. PMID 15054166.

Schwebke, J. R.; Burgess, D. (2004). "Trichomoniasis". Clinical Microbiology Reviews. 17 (4): 794–803, table of contents. doi:10.1128/CMR.17.4.794-803.2004. PMC 523559. PMID 15489349.

SUNEEL KUMAR VERMA
3rd Year 6th Semester (2021-2026)

Harpies

Herpes simplex virus (HSV), known as herpes, is a common infection that can cause painful blisters or ulcers. It primarily spreads by skin-to-skin contact. It is treatable but not curable.

Herpes simplex can cause both genital and oral herpes. Some people may experience sores during outbreaks while others may not have any symptom

CAUSE:

- You can transmit or contract HSV-1, or oral herpes, through direct contact with a herpes sore, saliva, or other bodily secretions during an episode
- Examples of direct contact include:
- • kissing
- • oral sex
- • other skin-to-skin contact
- As with HSV-1, you can transmit or contract HSV-2, or genital herpes, through direct contact with a herpes sore, saliva, or other bodily secretions during an episode.
- Direct contact might include:
- • kissing
- • oral sex
- • sharing sex toys during a sexual encounter
- • penetrative sex
- • other skin-to-skin contact at the infection site

MORTALITY RATE:

The mortality rate is approximately 70% in untreated patients and 19% in treated patients.

RISK FACTORS:

- Contact with genitals through oral, vaginal or anal sex.
- Having a partner who has the disease but is not taking medicine to treat it.
- Sexual contact with a person with HSV-1 or HSV-2.
- Presence or history of another sexually transmitted or blood-borne infection

COMPLICATIONS:

- Other sexually transmitted infections. Having genital sores raises your risk of giving or getting other STIs, including HIV/AIDS.
- Newborn infection. A baby can be infected with HSV during delivery
- Internal inflammatory disease these include the ureter, rectum, vagina, cervix and uterus.

- Infection of internal organs. Rarely, HSV in the bloodstream can cause infections of internal organs.

DIAGNOSIS:

- Diagnosis involves serological tests for syphilis, such as the rapid plasma reagin (RPR) or Venereal Disease Research Laboratory (VDRL) test, confirmed by treponemal-specific tests.
- Lumbar puncture is performed to analyze CSF for pleocytosis, elevated protein, and a positive VDRL test, which is highly specific for neurosyphilis.
- Swab Testing The gold standard for herpes diagnosis is a viral culture test or nucleic acid amplification test (NAT) of a sample of skin, crust, or fluid from a lesion.
- Herpes blood tests can detect antibodies to the herpes virus.
- IgM tests detect short-lasting antibodies that form to fight the virus shortly after an infection has occurred. IgG tests detect long-lasting antibodies that your body makes to fight the virus.
- The accuracy of various HSV-2 tests includes:9 HerpeSelect HSV-2 test: 99% sensitivity, 81% specificity Biokit HSV-2 Rapid Test: 84% sensitivity, 95% specificity.

TREATMENT:

There is no cure for genital herpes. Medicine can reduce symptoms and the chance of spreading it to others.

Medication antiviral medication, such as acyclovir, to prevent the virus from multiplying. Meanwhile, over-the-counter herpes treatments, often creams, can help manage tingling, itching, and pain.

Treatment for recurrent episodes is most effective when started within 48 hours of when symptoms begin. Antiviral medicines commonly given include acyclovir, famciclovir and valacyclovir.

REFERENCE:

https://www.healthline.com/

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SMRUTI LIPSA
3rd Year 6th-Semester -5 year program

Gonorrhea

EPIDEMOLOGY

In 2016, the World Health Organization estimated the global prevalence of T. vaginalis to be 5.0% in women and 0.6% in men. These data demonstrate a geographically variable burden of trichomoniasis among men, with prevalence ranging from 0.2% to 1.3%, with the highest rates occurring in Africa and the Americas . In the United States, T. vaginalis incidence among men was estimated to be 4.1 million infections in 2018 . Data from the 2013–2014 cycle of the National Health and Nutrition Examination Survey (NHANES) estimated the US T. vaginalis prevalence in men ages 18–59 years to be 0.5% compared with 1.8% among women . This study was the first to provide national prevalence data for T. vaginalis in US men. In addition, it was the first time NHANES utilized the highly sensitive and specific Hologic Gen-Probe Aptima T. vaginalis nucleic acid amplification test (NAAT) on urine specimens for diagnosis in both women and men . Given the sexually transmitted nature of T. vaginalis, the prevalence in men and women should be approximately equivalent.

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MUHAMMAD SHAHNAWAZ
9th semester 4th year Session 2020-25(MBBS)

Filariasis

Filariasis is caused by three species of nematodes (roundworms) that inhabit the lymphatics and subcutaneous tissues: Wuchereria bancrofti, Brugia malayi, and Brugia timori.

Transmission

After the bite of an infected mosquito, larvae enter the lymphatics and lymph nodes where they mature into white, thread-like adult worms. The adults live for 5 years, and females discharge microfilariae into the blood-stream, usually around midnight. In the South Pacific, the peak is less pro-nounced and occurs during the day.

Epidemiology

It is estimated that 120 million people are infected with these parasites.

• W. bancrofti occurs throughout the tropics and subtropics.

• B. malayi occurs mainly in South East Asia.

• B. timori is restricted to Timor in Indonesia.

Humans are the only host for W. bancrofti, but B. malayi has been found in felines and primates. Only a small proportion of people who are bitten by infected mosquitoes develop clinical disease.

Clinical Features

Most patients are asymptomatic, despite microfilaraemia.

• Acute infection may present with acute adenolymphangitis (ADL), acute dermatolymphangioadenitis (DLA), filiarial fever, and tropical pulmonary

eosinophilia.

• Chronic manifestations include lymphoedema (which may progress to elephantiasis) and hydrocele (which may be unilateral or bilateral).

• Renal involvement with discharge of lymph into the renal pelvis causes

chyluria, which can result in anaemia and hypoproteinaemia.

Complications

Complications

Occult filariasis - infection of other tissues without evidence in blood.[6] It can lead to:

Tropical pulmonary eosinophilia

Filarial arthritis

Filarial-associated immune complex glomerulonephritis

Filarial breast abscesses

Loiasis - co-endemic infection. It can cause severe drug interactions including encephalopathy if treated with ivermectin.

Diagnosis

Blood smear—a blood sample should be taken between 10 p.m. and 2a.m. (apart from if the patient is from the South Pacific when they may be taken in the daytime) and stained with Giemsa or Wright's stain.Microfilariae are occasionally seen in hydrocele fluid, chylous urine, or

lymph node aspirates.

• Circulating filarial antigen (CFA) assays are available for W. bancrofti infections and can be performed at any time of day.

• Serological tests may be positive but do not distinguish the different species, or current from past infection. Immunoassays and PCR-based

assays have been developed.

• Molecular tests—species-specific PCR tests are only available in the research setting.

• Ultrasonography of the lymphatic vessels in the spermatic cord may show motile adult worms.

Medical Treatment

Diethylcarbamazine (DEC) (6mg/kg/day for 12 days) is the treatment of choice for lymphatic filariasis. Side effects: fever,headache, nausea, arthralgia.

• Doxycycline (200mg/day for 4–6 weeks) has macrofilaricidal activity and reduces pathology in mild to moderate disease.

• Patients with concomitant infection with onchocerciasis should be treated with ivermectin (150 micrograms/kg stat), followed by standard treatment for filariasis.

• Patients with concomitant loiasis (E see Loa loa, pp. 556–7) and<2500 Loa loa microfilariae/mL of blood can be treated with DEC (8–10mg/kg/day for 21 days). Patients with

higher levels of Loa loamicrofilaraemia should be treated with doxycycline (200mg/day for4–6 weeks) or albendazole (200–400mg daily for 21 days).

• Mass drug treatment of populations in endemic areas use yearly single dose of DEC alone or in combination with albendazole or ivermectin.

Surgical treatment

Surgical treatments exist for the debulking of skin and creating lymphovenous anastomosis to improve drainage. Topical coumarin and flavonoids were shown to be effective in reducing lymphedema. It is thought that increased macrophage activity leads to the reuptake of proteinaceous material

Prevention

Mass drug administration to reduce the reservoir of microfilariae has been the cornerstone of prevention. Vector control using insecticide-treated bed nets has been useful in areas where anopheline mosquitoes transmit W. bancrofti. Travellers to endemic areas should be advised to avoid mosquito bites (protective clothing, insect repellent).

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Dr Ed Moran

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RITIK KUMAR

3rd Year 5th Sem (MBBS)

Meningitis

INTRODUCTION:-Meningitis is a life-threatening disorder that is most often caused by bacteria or viruses

ETIOLOGY:-Meningitis is defined as inflammation of the meninges. The meninges are the three membranes (the dura mater, arachnoid mater, and pia mater) that line the vertebral canal and skull enclosing the brain and spinal cord. Encephalitis, on the other hand, is inflammation of the brain itself.Meningitis can be caused by infectious and non-infectious processes (autoimmune disorders, cancer/paraneoplastic syndromes, drug reactions).The infectious etiologic agents of meningitis include bacteria, viruses, fungi, and less commonly parasites.

PATHOPHYSIOLOGY :-

Meningitis typically occurs through two routes of inoculation:-

•Hematogenous seeding-Bacteria colonize the nasopharynx and enter the bloodstream after mucosal invasion. Upon making their way to the subarachnoid space, the bacteria cross the blood-brain barrier, causing a direct inflammatory and immune-mediated reaction.

•Direct contiguous spread-Organisms can enter the cerebrospinal fluid (CSF) via neighboring anatomic structures (otitis media, sinusitis), foreign objects (medical devices, penetrating trauma), or during operative procedures.

RISK FACTORS:-

Age- children younger than 5 years

•Use of immuno suppressive drugs

• Chronic malnutrition

• AIDS

• CSF Shunt

• Chronic alcoholism

• Diabetes

• Chronic medical disorders (renal failure, diabetes, adrenal insufficiency, cystic fibrosis

DIAGNOSIS:-

•Meningitis is diagnosed through cerebrospinal fluid (CSF) analysis, which includes white blood cell count, glucose, protein, culture, and in some cases, polymerase chain reaction (PCR). CSF is obtained via a lumbar puncture (LP), and the opening pressure can be measured.

•Additional testing should be performed tailored on suspected etiology:

•Viral: Multiplex and specific PCRs

•Fungal: CSF fungal culture, India ink stain for *Cryptococcus*

•Mycobacterial: CSF Acid-fast bacilli smear and culture

•Syphilis: CSF VDRL

•Lyme disease: CSF *burgdorferi* antibody

TREATMENT:-

•Neonates - Up to 1 month old

Ampicillin intravenously (IV) and

Cefotaxime (or equivalent, usually ceftazidime or cefepime) IV or gentamicin IV and

Acyclovir IV

•More than 1 month old

Ampicillin IV and

Ceftriaxone IV

•Adults (18 to 49 years old)

Ceftriaxone IV and

Vancomycin IV

•Adults older than 50 years old and the immunocompromised

Ceftriaxone IV and

Vancomycin IV and

Ampicillin IV

•Meningitis associated with a foreign body (post-procedure, penetrating trauma)

Cefepime IV or ceftazidime IV or meropenem IV and

Vancomycin IV

•Meningitis with severe penicillin allergy

Moxifloxacin IV and

Vancomycin IV

•Fungal (Cryptococcal) meningitis

Amphotericin B IV and

Flucytosine by mouth

•**Antibiotics**

Ceftriaxone

Vancomycin

Ampicillin

Cefepime

Cefotaxime

REFERENCE:-1.Essentials of Medical [KD Tripathy]/ 2.Robbins Basic Pathology

HAFSA MUNIR
8th Semester 4th Year MBBS
Session 2020-2025

Acne

OVERVIEW:

Acne vulgaris is the most common cutaneous disorder. It affects more than 17 million Americans. Patients can experience significant psychological morbidity and rarely mortality due to suicide. Important that Physicians are familiar with Acne vulgaris and its treatment. Affects all races and ethnicities with equal significance. Darker skinned patients at increased risk for developing post-inflammatory hyperpigmentation and keloids.

DEFINITION:

Acne is an inflammatory disorder of the skin, which has sebaceous (oil) glands that connects to the hair follicle, which contains a fine hair.

In healthy skin, the sebaceous glands make sebum that empties onto the skin surface through the pore, which is an opening in the follicle. Keratinocytes, a type of skin cell, line the follicle. Normally as the body sheds skin cells, the keratinocytes rise to the surface of the skin. When someone has acne, the hair, sebum, and keratinocytes stick together inside the pore. This prevents the keratinocytes from shedding and keeps the sebum from reaching the surface of the skin. The mixture of oil and cells allows bacteria that normally live on the skin to grow in the plugged follicles and cause inflammation—swelling, redness, heat, and pain. When the wall of the plugged follicle breaks down, it spills the bacteria, skin cells, and sebum into nearby skin, creating lesions or pimples.

ETIOLOGY:

Role of Sebaceous Glands:

Our sebaceous (oil-producing) glands are affected by our hormones. In people who have acne, the glands are particularly sensitive, even to normal blood levels of these hormones. This causes the glands to produce too much oil. At the same time, the lining of the pores (the small holes in the skin's surface) becomes thickened and dead skin cells are not shed properly. A mixture of the oil (sebum) and dead skin cells builds up and plugs the pores producing blackheads and whiteheads. The plug of dead skin turns black from exposure to air and not due to dirt.

Bacterium:The acne bacteria (now known as ***Cutibacterium acnes***) live on everyone's skin, usually causing no problems. In those with acne, the build-up of oil creates an ideal environment for the bacteria to multiply. This is accompanied by inflammation which leads to the formation of red, swollen or pus-filled spots.

Medications:Sometimes acne can be caused by medication given for other health conditions or by certain contraceptive injections or pills. Some tablets taken by body-builders contain hormones that can trigger acne too.

Diet:Diet can influence acne. High glycemic index (GI) diets (e.g. sugar and sugary foods, white bread, potatoes, white rice etc.) have been shown to cause or aggravate acne. Switching to a low GI diet may lead to fewer spots. There is also some evidence that consuming milk and dairy products may trigger acne in some people, but this hasn't been studied in as much detail yet.

Level of Hormones:Most acne sufferers have normal hormone levels if tested; however, acne can sometimes be caused by a problem with the hormones. The most common problem with hormones is polycystic ovarian syndrome in females. If you are a woman and develop irregular periods, unusual hair growth or hair loss or other changes to your body, mention this to your doctor in case it is relevant.

PATHOGENESIS:

Excessive Circulating Androgens
↓
Enlargement and Hypersecretion of sebaceous glands
↓
Increased Production of Sebum
↓
Blockage of openings of Sebaceous glands
↓
Promotion of growth of Propionibacterium acnes
↓
Infection of Sebaceous Glands

TYPES OF ACNE:

Acne causes several types of lesions, or pimples. Doctors refer to enlarged or plugged hair follicles as comedones. Types of acne include:

- Whiteheads: Plugged hair follicles that stay beneath the skin and produce a white bump.
- Blackheads: Plugged follicles that reach the surface of the skin and open up. They look black on the skin surface because the air discolors the sebum, not because they are dirty.
- Papules: Inflamed lesions that usually appear as small, pink bumps on the skin and can be tender to the touch.
- Pustules or pimples: Papules topped by white or yellow pus-filled lesions that may be red at the base.

- Nodules: Large, painful solid lesions that are lodged deep within the skin.
- Severe nodular acne (sometimes called cystic acne): Deep, painful, pus-filled lesions.

TYPES OF ACNE ON BASIS OF SEVERITY:

There are Three levels of acne severity:

- Mild Acne: People with **mild acne** develop only a few noninflamed blackheads or whiteheads, or a moderate number of small, mildly irritated pimples. Pustules, which resemble pimples with yellow tops, may also develop. Blackheads appear as small flesh-colored bumps with a dark center. Whiteheads have a similar appearance but lack the dark center. Pimples are mildly uncomfortable and have a white center surrounded by a small area of reddened skin
- Moderate Acne: People with **moderate acne** have more blackheads, whiteheads, pimples, and pustules.
- Severe Acne: People with **severe acne** have either very large numbers of blackheads and whiteheads, pimples, and pustules or cystic (deep) acne. In cystic acne, cysts are large, red, painful, pus-filled nodules that may merge under the skin into larger, oozing abscesses.

SYMPTOMS:

Acne commonly appears on the face and shoulders. It may also occur on the trunk, arms, legs and buttocks. Skin Changes includes:

- Crusting of skin bumps
- Cysts
- Papules (Small Red Bumps)
- Pustules (Small Red Bumps Containing White or Yellow Pus)
- Redness around Skin Eruptions
- Scarring of the Skin
- Whiteheads
- Blackheads

DIAGNOSIS:

To diagnose acne, health care providers may:

- Ask about your family history, and, for girls or women, ask about their menstrual cycles.
- Ask you about your symptoms, including how long you have had acne.
- Ask what medications you are currently taking or recently stopped.
- Examine your skin to help determine the type of acne lesion.
- Order lab work to determine if another condition or medical disorder is causing the acne.

TREATMENT:

The goals of treatment are to help heal existing lesions, stop new lesions from forming, and prevent scarring. Medications can help stop some of the causes of acne from developing, such as abnormal clumping of cells in the follicles, high sebum levels, bacteria, and inflammation. Your doctor may recommend over-the-counter or prescription medications to take by mouth or apply to the skin.

Topical medications, which you apply to the skin, include:

- Over-the-counter products, such as benzoyl peroxide, which kills bacteria and may decrease the production of sebum.
- Antibiotics, which are usually used with other topical medications.

- Retinoids, which come from vitamin A and can help treat lesions and reduce inflammation. They can also help prevent the formation of acne and help with scarring.
- Salicylic acid, which helps break down blackheads and whiteheads and also helps reduce the shedding of cells lining the hair follicles.
- Sulfur, which helps break down blackheads and whiteheads.

For some people, the doctor may prescribe oral medications, such as:

- Antibiotics, which help slow or stop the growth of bacteria and reduce inflammation. Doctors usually prescribe antibiotics for moderate to severe acne, such as severe nodular acne (also called cystic acne).
- Isotretinoin, an oral retinoid, which works through the blood stream to help treat acne and open up the pore. This allows other medications, such as antibiotics, to enter the follicles and treat the acne. Similar to topical retinoids, taking the medication by mouth can also help prevent the formation of acne and help with scarring.
- Hormone therapy, used primarily in women, which helps stop the effects of androgens on the sebaceous gland.
- Corticosteroids, which help lower inflammation in severe acne, including severe nodular acne.

Some people who have severe acne or acne scarring that does not respond to topical or oral medications may need additional treatments, such as:

- Laser and other light therapies. However, researchers are still studying the best types of light and the amount needed to treat acne.
- Injecting corticosteroids directly into affected areas of your skin.
- Superficial chemical peels that a doctor recommends and applies to the area.
- Filling acne scars with a substance to improve their appearance.
- Treating acne scars with tiny needles to help induce healing.
- Surgical procedures to help treat and repair scarring.

PROGNOSIS:

Acne of any severity usually lessens spontaneously by the early to mid-20s, but some people, usually women, have acne into their 40s. Some adults develop mild, occasional, single acne lesions.

Mild acne usually heals without scars. Moderate to severe acne heals but often leaves scars. Acne can cause much emotional stress for adolescents and trigger social withdrawal. Counseling may sometimes be needed.

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ANKIT YADAV
5th sem 3rd Year(2021-2026/MBBS)

Osteoporosis

INTRODUCTION:-

metabolic skeletal disease characterized by low bone density and microarchitectural deterioration of bone tissue which results in increased bone fragility and susceptibility fracture The vertebrae, wrists ,and hips are the most common sites of fractures.

PATHOPHYSIOLOGY:-

Bone remodeling occurs throughout an individual's lifetime.
- In normal adults, the activity of osteoclasts (bone resorption) is balanced by that of osteoblasts (bone formation).
-normal bone remodeling in the adult result in gradually increase bone mass until the early 30s. with ageing the peak bone mas si gradualy decrease and
1. Calicitonin which inhibit bone resorption and promote bone formation. (decrease)
2.Estrogen which inhibit bone breakdown. (decrease)
3.PTH increase bone turnover and resorption. (increase)

RISK FACTORS:-

female more than male.
-increase age.
-inadequate intake of calcium and vit D. -estrogen deficiency or menopause. -family history.
- Lack of physical activity .
-Smoking, alcohol consumbtion
-medication. (corticosteroids, antiseizure)
- low weight and body mass index.

CAUSES:-

(1) Young adults
(2) Postmenopausal (type I)
(3) Senile (type lI)

SIGN AND SYMPTOMS:-

Osteoporosis has been called "silent disease" because bone mass is lost over many years with no sings or symptoms.
1.Loss of height.
2.Back pain.
3. vertebrae collapse. (dowager's hump)

DIAGNOSIS:-

1.x-ray studies: determine bone density.
2. radiographic: bone mass. (osteopenia). 3.ultrasonography:determine bone density.

4. Dual-energy x-ray absorptiometry

TREATMENT:-

1 calcitonin: a synthetic thyroid hormone usually prescribe
as a daily nasal spray to reduce factors that cause loss of calcium and increase reabsorption of calcium in the
gastrointestinal tract.

2. Selective Estrogen Receptor Modulators.

3. Hormone Replacement Therapy: ot increase serum estrogen levels, which in turn decrease the rate of bone resorption.

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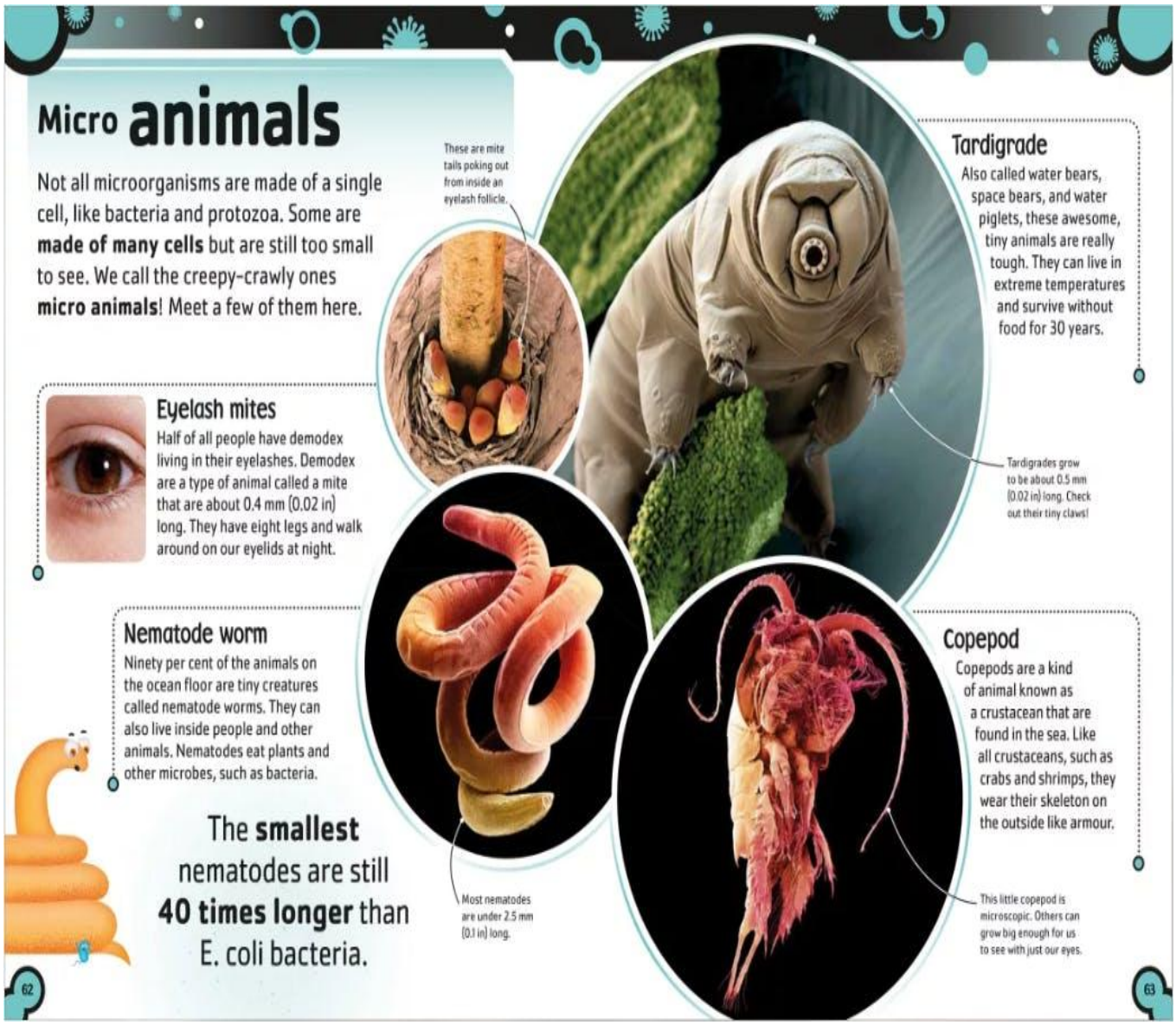

SIDDHANT KUMAR

5th sem 3rd Year (2021-2026/MBBS)

HIV

INTRODUCTION :-

HIV, which stands for Human Immunodeficiency Virus, is a virus that attacks the body's immune system, specifically targeting CD4 cells (also known as T cells), which are crucial for fighting off infections. Over time, HIV can weaken the immune system, making it harder for the body to fight infections and diseases. If left untreated, HIV can progress to AIDS (Acquired Immunodeficiency Syndrome), which is the final stage of HIV infection. However, with proper medical care, including antiretroviral therapy (ART), people with HIV can live long and healthy lives.

EPIDEMIOLOGY :-

•The epidemiology of HIV encompasses various aspects such as its global prevalence, distribution, transmission routes, affected populations, and trends over time. Global Prevalence: HIV/AIDS is a global pandemic, with the highest burden in sub-Saharan

RISK FACTOR:-

•Unprotected Sexual Intercourse

•Sharing Needles or breastfeeding

•Mother-to-Child Transmission

•Sexually Transmitted Infections (STIs)

•Blood Transfusions and Organ Transplants

SIGNS AND SYMPTOMS:-

•***Acute HIV Infection (Primary or Acute Retroviral Syndrome***

Swollen lymph nodes

Muscle aches and joint pain

Nausea, vomiting, or diarrhea

Night sweats

•***Chronic HIV Infection (Asymptomatic Stage):***

Many people with HIV may not experience any symptoms during this stage.

•***Advanced HIV Infection (Symptomatic HIV/AIDS):***

Persistent or recurrent fevers

Chronic diarrhea

Weight loss

Swollen lymph nodes

Oral thrush (white patches in the skin)

COMPLICATIONS :-

•***Opportunistic Infections***: HIV weakens the immune system, making individuals more susceptible to infections that would not normally cause illness in people with healthy immunity

•Neurological Complications

•HIV-Associated Cardiovascular Disease

•HIV-Associated Kidney Disease

•HIV-Associated Cancers

•Metabolic and Endocrine Disorders

•Mental Health and Substance Use Disorders

PATHOGENESIS OF HIV:-

•The pathogenesis of HIV involves the virus attacking the immune system, specifically CD4 cells, which are crucial for the body's defense against infections. HIV replicates inside these cells, leading to their destruction. This weakens the immune system, making the body vulnerable to opportunistic infections and cancers, ultimately progressing to AIDS if untreated.

DIAGNOSIS:-

•Diagnosing HIV typically involves testing for the presence of HIV antibodies or antigens in the blood. Common tests include ELISA, rapid antibody tests, and nucleic acid tests (NATs). If initial tests are positive, confirmatory tests like Western blot or PCR are usually conducted for verification.

TREATMENT:-

•The treatment of HIV typically involves antiretroviral therapy (ART), which consists of a combination of medications that suppress the HIV virus, reduce its replication, and slow down the progression of the disease. ART helps to prevent HIV from progressing to AIDS, reduces the risk of transmission, and allows people with HIV to live longer, healthier lives.

CONCLUSION:-

•HIV remains a significant global health concern, but advancements in diagnosis and treatment have transformed it into a manageable chronic condition rather than the fatal disease it once was. Early diagnosis through testing, followed by prompt initiation of antiretroviral therapy (ART), is crucial for controlling the virus and preventing its progression to AIDS.

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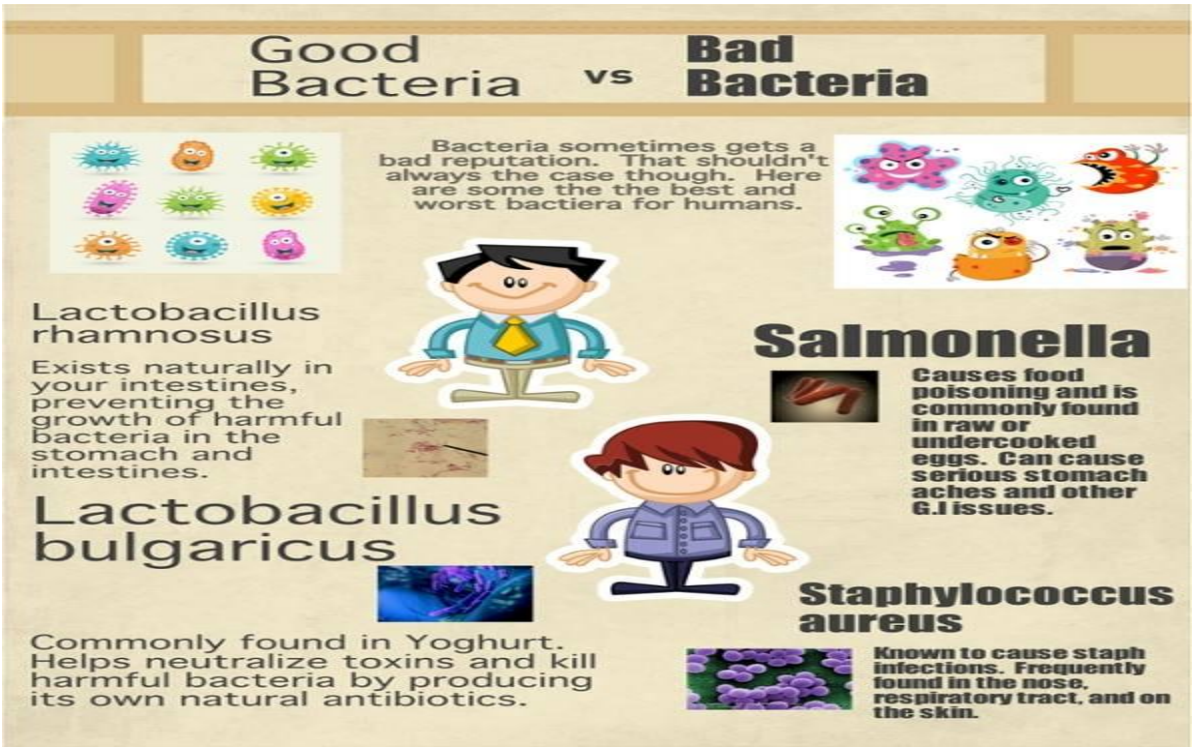

Tarnee Sahu--5th sem 3rd Year (2021-2026/MBBS)

Polio

Overview:-

Polio is short for "poliomyelitis." It's a virus that spreads easily between people who aren't vaccinated. If you get polio, you might have no symptoms or get flu-like symptoms. It’s less common, but the virus can make you very ill and cause you to lose the ability to move your limbs (paralysis). It could even kill you. People of any age who are not vaccinated can get polio, but kids under 5 have the highest risk.

Epidemiology:-

Cases due to wild poliovirus have decreased by over 99% since 1988, from an estimated 350 000 cases in more than 125 endemic countries, to just two endemic countries (as of October 2023). As long as a single child remains infected, children in all countries are at risk of contracting polio.

PATHOGENESIS:-

The virus enters via the fecal-oral or respiratory route, then multiplies in oropharyngeal and lower gastrointestinal tract mucosa. The virus is secreted into saliva and feces, from which it can be transmitted to others. The virus then enters the cervical and mesenteric lymph nodes.

SYMPTOMS:-

Symptoms depend on the type of polio you have Abortive polio symptoms:

Fever	Stomachache
Headache	Loss of appetite
Muscle aches	Nausea
Sore throat	Vomiting

RISK FACTORS:-

You're at risk of getting polio if you're unvaccinated. If you have been vaccinated, it is extremely rare for you to get it. Children under 5 are at higher risk of infection until they are fully vaccinated. You are also at risk if you are around someone who has polio or if you travel to a country where polio exists.

DIAGNOSIS:-

Poliovirus can be detected in specimens from the throat, feces (stool), and occasionally cerebrospinal fluid (CSF) by isolating the virus in cell culture or by detecting the virus by polymerase chain reaction (PCR).

TREATMENT:-

There's no cure for polio. If you have it, your doctor will focus on making sure that you're comfortable and try to prevent other health issues. Some treatments include:

-Pain relievers (like ibuprofen)

-A ventilator (a device that helps you breathe)

-Physical therapy that can help keep your muscles working

-Bed rest and fluids for flu-like symptoms

-Antispasmodic medications to relax muscles

-Antibiotics for urinary tract infections

-A heating pad for muscle aches and spasms

-Corrective braces

-Pulmonary rehabilitation to help with lung complications

COMPLICATIONS:-

The most significant complications of polio infection include paralysis with bulbar involvement, fatal respiratory and cardiovascular collapse, and postpolio syndrome; postpolio syndrome is characterized by new-onset or progressive muscle weakness in a patient previously diagnosed with poliomyelitis.

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VIVEKANAND YADAV

5th Sem 3rd Year (2021-2026/MBBS)

Hepatitis- C

OVERVIEW :-

Hepatitis C is a viral infection that causes liver swelling, called inflammation. Hepatitis C can lead to serious liver damage. The hepatitis C virus (HCV) spreads through contact with blood that has the virus in it.

EPIDIMOLOGY:-

Globally, an estimated 50 million people have chronic hepatitis C virus infection, with about 1.0 million new infections occurring per year. WHO estimated that in 2022, approximately 242 000 people died from hepatitis C, mostly from cirrhosis and hepatocellular carcinoma (primary liver cancer).

MANAGEMENT :-

Antiviral medications, including sofosbuvir and daclatasvir, are used to treat hepatitis C. Some people's immune system can fight the infection on their own and new infections do not always need treatment. Treatment is always needed for chronic hepatitis C.

National Guidelines for the Management of Viral Hepatitis

Viral hepatitis is defined as inflammation of the liver cells due to viral infection. The burden of liver disease in South Africa is mostly underestimated as viral hepatitis, in particular chronic infection, is a silent and neglected cause of morbidity and mortality. However, the burden of disease is likely substantial given the prevalence of chronic viral hepatitis. This burden is further compounded by the lack of screening and access to care and treatment as well as inadequate disease surveillance, human and financial resources. The National Guidelines for the Management of Viral Hepatitis were developed, with the purpose to: inform healthcare workers in the public and private sectors about the disease, its epidemiology in South Africa and current methods of diagnosis and therapy strengthen the healthcare response to viral hepatitis empower communicable diseases workers and stakeholders to make informed decisions regarding appropriate and cost effective interventions

PATHOGENESIS:-

HCV is a non-cytopathic virus[35] that enters the liver cell and undergoes replication simultaneously causing cell necrosis by several mechanisms including immune-mediated cytolysis in addition to various other phenomena such as hepatic steatosis, oxidative stress and insulin resistance.

DIAGNOSIS:-

HCV-people who infection is diagnosed in 2 steps: Testing for anti-HCV antibodies with a serological test identifies have been infected with the virus. If the test is positive for anti-HCV antibodies, a nucleic acid test for HCV ribonucleic acid (RNA) is needed to confirm chronic infection and the need for treatment.

TREATMENT:-

Hepatitis C is treated using direct-acting antiviral (DAA) tablets.DAA tablets are the safest and most effective medicines for treating hepatitis C.They're highly effective at clearing the infection in more than 90% of people.The tablets are taken for 8 to 12 weeks. The length of treatment will depend on which type of hepatitis C you have.Some types of hepatitis C can be treated using more than 1 type of DAA. NHS- approved hepatitis C medicines include:

sofosbuvir

a combination of ledipasvir and sofosbuvir

a combination of ombitasvir, paritaprevir and ritonavir, taken with or without dasabuvir

a combination of elbasvir and grazoprevir

a combination of sofosbuvir and velpatasvir

a combination of sofosbuvir, velpatasvir and voxilaprevir

a combination of glecaprevir and pibrentasvir

Ribavi

COMPLICATION:-

Chronic hepatitis C can be a serious disease resulting in long-term health problems, including liver damage, liver failure, cirrhosis, liver cancer, and even death. It is the most common reason for liver transplantation in the United States.

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UMAR GUL RIAZ
8th Semester 4th Year MBBS Session

Cellulites

OVERVIEW: Cellulitis is a common bacterial skin infection, with over 14 million cases occurring in the United States annually. This activity educates the learner on the etiology, epidemiology, evaluation, and treatment of cellulitis. It provides the latest updates on how to accurately diagnose, effectively treat, and manage patients with bacterial cellulitis. Upon completing the activity, the learner should be able to differentiate cellulitis from other mimickers correctly. The learner will know how to discern when cellulitis treatment is appropriate in the outpatient setting with oral antibiotics versus when a patient should be hospitalized and treated with intravenous antibiotics, and how the interprofessional team can best manage patients with cellulitis.

INTRODUCTION TO CELLULITIS: Cellulitis is a common bacterial skin infection, with over 14 million cases occurring in the United States annually. It accounts for approximately 3.7 billion dollars in ambulatory care costs and 650000 hospitalizations annually.

Cellulitis typically presents as a poorly demarcated, warm, erythematous area with associated edema and tenderness to palpation. It is an acute bacterial infection causing inflammation of the deep dermis and surrounding subcutaneous tissue. The infection is without an abscess or purulent discharge.

Beta-hemolytic streptococci typically cause cellulitis, generally group A streptococcus **(i.e., *Streptococcus pyogenes*)**, followed by methicillin-sensitive ***Staphylococcus aureus***. Patients who are immunocompromised, colonized with methicillin-resistant Staphylococcus aureus, bitten by animals, or have comorbidities such as diabetes mellitus may become infected with other bacteria.

If the clinician correctly identifies and promptly treats cellulitis, it typically resolves with appropriate antibiotic treatment.

ETIOLOGY: Cellulitis is usually caused when bacteria enter a wound or area where there is no skin. The most common bacteria that cause cellulitis include:

- Group A ß - hemolytic streptococcus (Strep)
- Streptococcus pneumoniae (Strep)
- Staphylococcus aureus (Staph)

Staph and strep bacteria are commonly found on the skin and mucous membranes of the mouth and nose in healthy people. The infection happens when there is a break in the skin that allows

the bacteria to enter. Other causes may include human or animal bites, or injuries that happen in water.

EPIDEMIOLOGY: Cellulitis is relatively common, and most often occurs in middle-aged and older adults. When comparing men and women, there is no statistically significant difference in the incidence of cellulitis. There are approximately 50 cases per 1000 patient-years.

PATHOPHYSIOLOGY: Cellulitis is characterized by erythema, warmth, edema, and tenderness to palpation resulting from cytokine and neutrophil response from bacteria breaching the epidermis. The cytokines and neutrophils are recruited to the affected area after bacteria have penetrated the skin leading to an epidermal response. This response includes the production of antimicrobial peptides and keratinocyte proliferation and is postulated to produce the characteristic exam findings in cellulitis.

Group A Streptococci, the most common bacteria to cause cellulitis, can also produce virulence factors such as pyrogenic exotoxins (A, B, C, and F) and streptococcal superantigen that can lead to a more pronounced and invasive disease.

TYPES: Cellulitis can have different names and characteristics depending on where it develops in your body.

- **Buccal cellulitis**
  - Buccal cellulitis develops on your cheek. This most often happens to very young children under a year old. It sometimes occurs alongside a tooth problem.
- **Perianal streptococcal cellulitis**
  - Perianal streptococcal cellulitis develops in and around your anal and rectal areas. This type occurs most often in children.
- **Pre-septal cellulitis**
  - Pre-septal cellulitis develops in your eyelid and at the front of the eye and is most common in children under five years old. It can cause swelling, redness, warmth, and pain in the upper and lower eyelids and other areas near the eye.
- **Orbital cellulitis**
  - Orbital cellulitis develops behind the eye, and the eye sometimes looks normal even when the tissues behind it are infected. The eyeball may turn red or bulge out, you may have trouble moving it or seeing out of it, and your upper and lower eyelids may swell, change color, or change texture. This is less common and often more dangerous than pre-septal cellulitis.

SYMPTOMS:

- The most common symptom of cellulitis is:
- Dull pain or tenderness in the area of skin involvement.
- Other cellulitis symptoms can include:
- Swelling, warmth, and redness in a distinct area of skin.
- These symptoms commonly worsen, and the redness may expand over the course of hours to days.
- The onset of cellulitis may be gradual or sudden.
- Itching is not a typical symptom of cellulitis.

- The skin is usually smooth and shiny rather than raised or bumpy.
- However, occasionally in cases of cellulitis, blisters or small pimples may form in the skin.
- In more severe cases, fever and chills can develop.
- If cellulitis is affecting the patient's lower extremities, careful evaluation should be made to look between the patient's toes for fissuring or tinea pedis.
- Additionally, it can affect the lymphatic system and cause underlying lymphadenopathy.
- The associated edema with cellulitis can lead to the formation of vesicles, bullae, and edema surrounding hair follicles leading to Peau d'orange.

RISK FACTORS:

- Those conditions include having a:
- Weakened immune system
- Having a history of cellulitis
- Other skin problems (such as psoriasis or eczema)
- Being obese or overweight
- Lymphedema
- Use of illicit injectable drugs

DIAGNOSIS:

- To diagnose cellulitis, your healthcare provider will ask about your symptoms and perform a physical examination of the affected area.
- Some Additional test that confirms the diagnosis:
- Blood Test: A Blood Test will confirm whether the cellulitis infection has spread to your blood.
- Skin Test: A skin test will identify the type of bacteria responsible for your cellulitis, which helps your healthcare provider prescribe the most appropriate antibiotic.
- Bacterial Culture: A bacterial culture will identify the type of bacteria responsible for your cellulitis.

TREATMENT:

- Patients presenting with mild cellulitis and displaying no systemic signs of infection should be covered with antibiotics that target the treatment of streptococcal species. Though believed to be a less common cause, consider coverage of MRSA.
- The duration of oral antibiotic therapy should be for a minimum duration of 5 days.
- In non-purulent cellulitis, patients should receive cephalexin 500 mg every 6 hours.
- If they have a severe allergic reaction to beta-lactamase inhibitors, treat with clindamycin 300 mg to 450 mg every 6 hours.
- In patients with purulent cellulitis, methicillin-resistant staph aureus colonization, cellulitis associated with an abscess or extensive puncture wounds, or a history of intravenous drug use, patients should receive antibiotics that cover against methicillin-resistant staph aureus as well.
- Cellulitis with MRSA risk factors should be treated with trimethoprim-sulfamethoxazole 800 mg/160 mg twice daily for 5 days in addition to cephalexin 500 mg every 6 hours.
- If a patient has an allergy to trimethoprim-sulfamethoxazole, treat with clindamycin 300 mg to 450 mg every 6 hours.

- A longer duration of antibiotic treatment may be a consideration in patients who show minimal improvement with antibiotic therapy within 48 hours.

COMPLICATIONS: Sometimes, cellulitis will spread. As this happens, the redness and inflammation will travel across your limb or other body part. This is a very bad sign which means the infection is growing. If you don't treat cellulitis, it could develop into other more severe complications, such as:

- Abscesses
- Bone infection (osteomyelitis)
- Gangrene (tissue death)
- Infection of the heart (endocarditis)
- Inflammation of the lymph vessels (lymphangitis)
- Meningitis
- Shock

PROGNOSIS: If the clinician promptly identifies cellulitis and initiates treatment with the correct antibiotic, patients can expect to notice an improvement in signs and symptoms within 48 hours. Annual recurrence of cellulitis occurs in about 8 to 20% of patients, with overall reoccurrence rates reaching as high as 49%. Recurrence is preventable with prompt treatment of cuts or abrasions, proper hand hygiene, as well as effectively treating any underlying comorbidities. There is approximately an 18% failure rate with initial antibiotic treatment. Overall, cellulitis has a good prognosis.

PREVENTION:

- Cleaning your wounds or sores with antibacterial soap and water.
- Applying an antibiotic ointment on your wounds or sores.
- Covering your wounds or sores with a bandage to prevent dirt or bacteria from entering the area.
- Refraining from touching or rubbing your affected areas.
- Getting medical attention right away for any deep cuts or puncture wounds.

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ROHA RAJAB

4th Year-8th Semester MBBS (Session 2020-2025)

Dermatitis

DEFINATION:

- "Dermatitis" simply means inflammation of the skin and the term is used interchangeably with "eczema", defined as an inflammatory process of the skin characterised variably by erythema, oedema, vesiculation, scaling, fissuring and lichenification, depending on severity and chronicity"
- Eczema is not a single disease entity, but describes a pattern of inflammatory responses originating in the dermis. The inflammation may be acute, subacute or chronic, each representing one stage in the evolution of the inflammatory process.
- Dermatitis may usefully be divided into endogenous and exogenous types, depending on whether the main precipitating factor of the inflammatory response is within the body or is caused by some external agent. There is no absolute division, however, and in many cases, the skin condition may be due to an interaction between external agents and individual responses of the body. In addition, endogenous and exogenous types may be present in the same patient.

CLINICAL FEATURES:

1. **Endogenous eczema:**

Atopic eczema is particularly common in children, but occurs at all ages. It may occur all over the body but especially affects the flexures, appearing as areas of red and scaly skin which may become lichenified. It tends to be associated with intense itching and, consequently, the prevention of scratching is a major problem. Atopic hand eczema is a common manifestation of adult atopic dermatitis.

Asteatotic eczema (eczema craquele) appears when there is excessive drying of the skin. It is most commonly seen on the front and side of the legs, which become dry and scaly. If there is further drying accompanied by scratching, red plaques appear with long horizontal fissures. If the condition worsens, the horizontal fissures are joined together by shorter vertical fissures and the whole area resembles cracked porcelain.

Seborrhoeic dermatitis most commonly occurs on the scalp, face (especially the nasolabial folds and eyebrows) and presternal area of skin. Dandruff may be considered a banal and minor form of the condition. In more severe forms it is characterised by dull or yellowish greasy scales.

Exogenous eczemia: Exogenous eczema has well-defined external triggers, and an inherited tendency plays only a small part.

- **Irritant contact dermatitis** is the most common type of eczema seen affecting the hand. It is typically seen in those engaged in 'wet work' and people whose occupations involve them in repeated wetting of the hands or exposure to irritant chemicals. The initial changes are dryness, chapping and redness. Cracking and fissuring may then occur. The changes at this stage are subacute. If there is further irritation, the itching intensifies and excoriation occurs. Superadded infection may be present.
- **Allergic contact dermatitis** occurs when a specific allergy develops to some substance with which the skin is in contact. As with any type of eczema, the condition can be exacerbated by exposure to washing, scratching, further irritants, and infection, or even by medication.

STAGES OF ECZEMIA:

- **Acute eczema** is characterised by erythema and a moderate to intense degree of inflammation. There is intense itching accompanied by an extreme desire to scratch. In all but very mild cases, some vesicle formation and blistering occurs. If no further exposure occurs, acute eczema improves spontaneously, passing through the subacute stage as it resolves.
- **Subacute eczema** is characterised primarily by erythema and scaling, usually with indistinct borders. The intensity of itching varies greatly. Subacute inflammation may be the first manifestation of eczema, or it may follow acute eczema. Equally, acute eczema may follow the subacute form if it becomes infected or irritated. Subacute eczema will resolve completely without scarring if the causes are removed. If external irritation or excoriation continues, this stage may evolve into chronic eczema. Subacute eczema is seen in conditions such as allergic contact dermatitis, asteatotic eczema, atopic dermatitis, nappy rash, exposure to chemicals, irritant contact dermatitis, nummular eczema and stasis dermatitis .
- **Chronic eczema** results from uncontrolled scratching and/or continuing irritation. There is thickening and fissuring of the inflamed area, most frequently seen in the areas of the body which are easily reached. The itching is always at least moderate in intensity. Scratching, which often occurs during sleep as well as during waking hours, causes excoriation. The condition may become self-perpetuating since, as it becomes worse, the itching becomes more intense.

  Aetiology:

Endogenous Eczema

Atopic eczema is genetically determined but may be exacerbated by exposure to irritant chemicals and by scratching.

- **Seborrhoeic dermatitis** is caused by infection of the skin with the yeast Malassezia furfur
- **Stasis dermatitis** probably has a number of different contributing causes. The basic cause is always related to excess hydrostatic pressure in the venous system. The variants depend on local anatomical and physiological factors.

Exogenous eczema

- **Irritant contact dermatitis**. The most common irritants are solvents and detergents. Dermatitis occurs more often in people who wash and dry their hands frequently e.g. hairdressers, health care workers, catering staff.
- **Allergic contact dermatitis** can be caused by exposure to many different agents. It is manifestation of a delayed hypersensitivity reaction occurring on the skin of a previously sensitised individual.
- **Occupational dermatitis** is a skin condition which may originate from occupational exposure, but is often influenced by many other factors. There can be a constitutional predisposition such as previous childhood eczema, previous sensitisation to specific allergens, development of allergic reactions to topical medicaments used to treat the condition.

DIAGNOSIS:

SKIN BIOPSY At times, a dermatologist needs to remove a small piece of skin for lab testing. This procedure, called a biopsy, is usually only necessary if doctors have not been able to diagnose your condition during a physical exam or patch test. A skin biopsy is a minor procedure performed in a doctor's office. The doctor typically injects a local anesthetic to numb the skin and uses a scalpel, a sharp blade, or punch instrument to remove a small section of the rash. The biopsied area is covered by a bandage and heals within a week.

TREATMENT: The treatment for dermatitis varies, depending on the cause and your symptoms. If home care steps don't ease your symptoms, your doctor may prescribe medicine. Possible treatments include: Applying to the rash a prescription-strength corticosteroid cream, gel or ointment

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MISBAH ABBAS

4th Year-8th Semester MBBS (Session 2020-2025)

Skin Bacterial Infection

Introduction:

Skin infections can be caused by bacteria (often Staphylococcal or Streptococcal) either invading normal skin, or affecting a compromised skin barrier (e.g, skin affected by atopic dermatitis, or surgical wound sites). • The skin is our first line of protection against the environment. A local or systemic response is activated when this protective barrier is invaded. Microorganisms that invade the skin can be part of the external environment or the normal skin microbiome.

Occurrence:

- Bacterial skin infections are a common reason for emergency visits. Children under five years and adults over 65 years old are affected more often than other age groups. In 2005, the World Health Organization (WHO) reported a high prevalence of skin disease in children from developing countries in sub-Saharan Africa.
- Gender may also play a role; in one North American study, men comprised 60-70% of all cases of cellulitis.

Route of Infection:

• Skin (pores , hair follicles)

• Wounds (burns , scratches , cuts)

• Insect and animal bites

Types of Infections:

- **Primary Infection:**
  • Caused by a single pathogen , usually effect normal skin
  • Impetigo, folliculitis and boils are common types
  • Most common primary skin pathogens are S Aureus ,beta .hemolytic streptococcus and coryneform bacteria .
  • Organisms usually enter through a break in a skin .
- **Secondary Infection:**

• Secondary infections occur in skin that is already disease .

• Because of the underlying disease , the clinical picture and course of these infections vary.

• Intertrigo and toe web infection are examples.

Common skin infections include:

• Impetigo • Folliculitis • Cellulitis • Carbuncle • Furuncle • Erysipelas

Impetigo

• Impetigo is a bacterial infection that affects the skin, causing sores and blisters .

• Appears as a honey color crust .

• Impetigo is a common contagious skin infection. Bacteria like Staphylococcus aureus or Streptococcus pyogenes infect the outer layers of your skin , causing blisters and sores .

Two types

• Bollous • Non bollous

Folliculitis:

• Folliculitis is a common skin condition that happens when hair follicles become inflamed.

• It is caused by staphylococcus aureus or sometimes by pseudomonas aeruginosa.

• The bacteria is commonly found in contaminated whirl pools ,hot tubs and physiotherapy pools.

• Children gets hot tub folliculitis More .

• Hot tub folliculitis occur because of inadequate treatment of water with chlorine or bromine

Cellulitis:

• It is an acute bacterial infection of the skin . Affect the middle layer of the skin (dermis) and the tissues below.

• Most often caused by streptococcus and staphylococcus.

• Cellulitis can occur anywhere on the body (in adults it often occurs on the legs face or arms , in children it most common on face or around the anus) .

• An infection on the face could lead to dangerous eye infection.

Carbuncle and Furuncle:

• Carbuncle are clusters of furuncles connected subcutaneously causing deeper suppuration and scarring .They are smaller and more superficial then subcutaneous abscess.

• Furuncle are skin abscess caused by staphylococcus infection which involve hair follicle and surrounding tissue . Deep seated inflammatory nodule with pustular center that develops around hair follicle which are painful , localized and abscessed .

Laboratory diagnosis of bacterial infections of skin

Specimen collection

1.Skin biopsy
2.Skin swab
3.Pus swab
when pustules or vesicles are present , the roof or crust is removed with sterile surgical blade . Pus or exudate is spread as thinly as possible on a clear glass slide for gram staining .
1 . Specimen is Gram stained. gram (+ve) cocci in cluster or chains appears .

2. Culture

Use blood agar , Mannitol salt agar , for identification of bacteria . On blood agar : both S.aureus , Strept. Pyogen produce beta hemolysis. Mannitol salt agar : high salt inhibits the growth of other organisms , S.aureus ferments mannitol, the acid produced turns the colonies yellow .

Treatment:

• Local therapy

– cleaning with soap water and weak KMNO4 solution

- Removal of crust with KMNO4 solution

- Application of antibacterial creams or ointments

• Systemic therapy

– Antibiotics
• Erythromycin
• Ciprofloxacin
• Amoxicillin

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MUHAMMAD YASIR MAJEED

4th Year-8th Semester MBBS (Session 2020-2025)

Pyrexia of Unknown Origin (PUO)

DEFINATION:

A persistent fever above 38.3°C (100°F) that evades diagnosis for at least 3 weeks, including 1 week of investigation in hospital.

The classic criteria for adult PUO are: Fever of 38.3°C (101°F) or greater lasting for at least three weeks. No identified cause after three days of hospital evaluation or three or more outpatient visits. Fever is a response to cytokines and acute phase proteins occurs in infections and in noninfectious condition.

ETIOLOGY:

3 common etiologies which account for the majority of classic PUO:

1. Infections
2. Malignancies
3. Collagen Vascular Disease

Others/Miscellaneous which includes drug-Induced fever.

1.Infections:

Bacterial: Abscesses, TB, complicated UTI, endocarditis, osteomyelitis, sinusitis, Lyme disease, prostatitis, cholecystitis,empyema, biliary tract infection, brucellosis, typhoid,leptospirosis, Q Fever, borreliosis, etc.

Parasite: Malaria, toxoplamosis, leishmaniasis, etc.e Fungal: histoplasmosis, etc.

Viral: CMV, infectious mononucleosis,, HIV, etc.

2.Malignancies:

Haematological like leukemias ,lymphomas and myeloma or solid malignancy like renal ,hepatic ,colon stomach and pancreas.

3.Collagen Vascular Disease:

like cardiovascular diseases atrial myxoma ,aortitis . respiratory diseases like extrinsic allergic alveolitis , pulmonary embolism and sarcoidosis . Gastrointestinal diseases like inflammatory

bowel disease , chronic liver diseases . Endocrine and metabolic diseases like thyrotoxicosis ,thyroiditis Addison disease . haematological diseases like autoimmune haemolytic anemia , graft versus host disease and thrombotic thrombocytopenic purpura . Inherited like familial Mediterranean fever and familial fever syndrome . drug fever due to hypersensitivity reaction or antibiotic fever . Factitious fever .

Pathophysiology of fever:

Temperature of the body is controlled by core body temperature which is controlled by and in the anterior hypothalamus (thermo-regulator) , so fever is elevation of the temperature of the core body temperature (normally 36.5 – 37.2) . Temperature > 42 or < 35 ℃ is fatal .

Fever occur when foreign body enter the body like bacteria ,virus ,fungal or malignant cell ,inflammation …etc (pyrogen)stimulate immune system to stimulate prostaglandin E2 where it affect the hypothalamus to increase body temperature .

Body temperature is examined by thermometer in the mouth which is near the normal core temperature , or rectal exam where it more than mouth temperature by 0.5 degree so we should reduce 0.5 degree from the reading result . or by axillary exam where it less than mouth by 0.5 degree so we should add 0.5 degree to the reading degree .

Types of Fever:

a) **continuous Fever :** Temperature remains above normal throughout the day and does not fluctuate more than 1 °C in 24 hours, e.g. lobar pneumonia, typhoid, meningitis, urinary tract infection, brucellosis, or typhus .
b) **Remittent Fever:** Temperature remains above normal throughout the day and fluctuates more than1 °C in 24 hours, e.g., infective endocarditis.
c) **Intermittent Fever:** The temperature elevation is present only for a certain period, later cycling back to normal, e.g. malaria, kala-azar, pyaemia, or septicemia .

Classification of Fever:

Normal 36.5 -37.2 ℃ , hypothermia < 35℃ , fever > 37.2 ℃ , grade low fever 37.3 -38℃ , hyperpyrexia > 40 ℃ .

Clinical Assesment:

Proper history including travels ,drug use exposure to noxious materials family and inherited diseases ..etc and full examination must be done usually young patients have infectious causes while elderly patients may have infectious and non infectious causes detailed history and examination must be repeated at regular interval to detect any new signs indicate vasculitis or endocarditis in men we should care about hidden area including prostate and breast in female and always we should look for factitious fever in which the patient looks well and his temperature > 41 ℃ lose of diurnal variation of temperature lose of fever pulse rate relation and no sweating during defervescence , presence of self induce harm normal temperature during observation at hospital .

Investigation:

Patients should be sent for complete blood count and ESR with C-reactive protein , renal function test RFT , liver function test LFT urinalysis and general stool exam ,blood for culture and sensitivity , radiological study according to need like chest X-ray ,organ suspected affected CT-scan if CNS involvement suspected CSF study cerebrospinal fluid study , infection diagnosis if suspected by nucleic acid amplification serological test ,splenic aspiration for leishmaniasis ,radiolabel led white cell study or positron emission tomography (PET-sacn) for detection of hidden infection site . For connective tissue diagnosis by radiological study , antibody studies . more invasive investigation may needed like liver biopsy if there are elevated liver enzyme or radiological indications , bone marrow aspirate and biopsy used for microscopic study ,histopathological study and culture for microbiological study , blind organ if no abnormal markers indicate for biopsy are unhelpful ,any lymphadenopathy must studied by biopsy ,laparoscopy and organ biopsy may be needed .

Treatment:

According to the cause specific antibiotics for infectious causes , steroid for immunological and connective tissue causes and chemotherapy for malignancy .

Prognosis: Mortality rate 30-40% in malignant conditions in elderly patients . While if long term fever without diagnosis usually the prognosis is good .

PUO in immunocompromised patients:

These patients are either congenital causes ,on immunosuppressant drugs ,on corticosteroids medication ,malignant diseases . the patient has neutropenia , lymphopenia , monocytopenia or mixed this lead to ordinary and opportunistic infection in these cases the diagnosis of infection should be rapid because within hours might enter into septicemia and septic shock which carry high mortality rate. The clinical features in these patients are atypical the fever may be absent due to low immunological response, opportunistic infection which not occur in immunocompetant patient must be diagnosed and microbial resistance increase due to previous use of antibiotics . The fever in these patients should be differentiated from primary disease , opportunistic or new infection or drug fever .

PUO in HIV patients:

This is due acute seroconversion or AIDS defining condition and it will be studied in the relevant subject.

PUO due to nosocomial infection:

This occur in patients after admission to hospital from central venous catheter ,double lumen venous catheter , ventilator …etc . The bacteria usually meticillin resistant staphylococcus aureus MRSA or coagulase negative staphylococci ,enterococci , gram , s negative bacteria or fungal infection . It proved by blood culture or instrument tip culture . Treated by removing medical instrument and antibiotics against specific bacteria for minimum 2 weeks .

Reference:

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ABDULLAH SHARIF 4th Year-8th Semester MBBS (Session 2020-2025)

Fungal Infection

OVERVIEW

Fungal infections are any disease or condition that you get from fugus. They usually affect your skin, hair, nails or mucous membranes but they can also infect your lungs or other parts of your body. You're at higher risk for fungal infections if you have a weakened immune system.

EPIDEMOLOGY

Global estimates found:30,00,000 cases of Chronic Pulmonary Asperigillosis
2,23,000 cases of Cryptococcal Meningitis
7,00,000 cases of Invasive Candiasis
5,00,000 cases of Pneumocystitis jerovecii Pneumonia
2,50,000 cases of Invasive Asperigillosis
1,00,000 cases of Disseminated Histoplasmosis
10,00,000 cases of Fungal Asthma
10,00,000 cases of Fungal Keratitis
In India, 13,80,000 lakh people are affected with bronchopulmonary asperigillosis

CLASSIFICATION

Fungal infections are classified into 4 types:

1. Superficial Infections
2. Cutaneous Infections
3. Subcutenous Infections
4. Systemic Infections

SYMPTOMS

Symptoms of fungal infections can range from mild to severe.Some symptoms are as follow:

1. Asthama like symptoms
2. Fatigue
3. Headache
4. Night Sweats
5. Weight loss
6. Chest pain
7. Muscle ache
8. Joint pain
9. Itchy or scaly skin

RISK FACTORS

Risk factors of fungal infections include:

1. Dampness and humidity
2. Menopausal status

3. Poor blood circulation
4. Suppressed immune system
5. Nail and skin injury
6. Other infections

COMPLICATIONS

Complications of fungal infections are given below:

1. Endocarditis
2. Meningitis
3. Encephilitis
4. Liver abscess
5. Septic arthritis
6. Splenic abscess
7. Renal bladder abscess
8. Osteomylitis

DIAGNOSIS

1. Clinical Presentation
2. Microscopic Examination
3. Culture Test
4. Lab Tests

TREATMENT

Fungal infections are typically treated with antifungal medications, usually with ones that are applied directly to the affected area (called topical medications). Topical medications may include creams, gels, lotions, solutions, or shampoos.

Antifungal medications may also be taken by mouth.

In addition to medications, people may use measures to keep the affected areas dry, such as applying powders or wearing open-toed shoes.

Corticosteroids can help relieve inflammation and itching caused by some infections, but these should be used only when prescribed by a doctor.

Common names for antifungal medicines include:

- clotrimazole.
- econazole.
- miconazole.
- terbinafine.
- fluconazole.
- ketoconazole.
- amphotericin.

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SHAHID KHAN
4th Year 8th Semester
MBBS (Session 2020-2025)

COVID-19

Definition:

Coronaviruses are a group of related viruses that causes disease in mammals and birds.In humans, this virus causes respiratory tract infections .

Realm- Riboviria

Phylum- incertae sedis

Order- Nidovirales

Family- Coronaviridae

Subfamily- Orthocoronavirinae

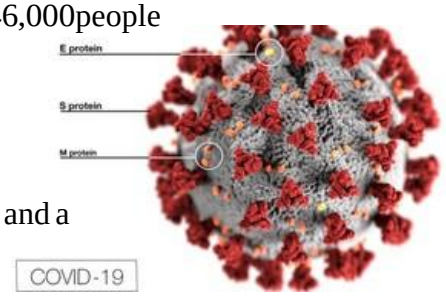
Pandemic:

This virus has been declared by WHO (World Health Organization) as pandemic.

Pandemic can be used for a disease that has spread across an entire country or other largelandmass.

Few months ago no one knew that a virus named SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) even existed.

Till April 2020, this virus spreaded to almost every country, infecting at least 446,000people whom we know about and many more whom we don't.



Structure of coronavirus:

They are enveloped viruses with a positive-sense single-stranded RNA genome and a nucleocapsid of helical symmetry.

They have got characteristic club-shaped spikes that project from their surface.Average diameter of virus particle is 120 nm.

Spikes are almost of 20 nm while the diameter of the envelope is about 80 nm.

The viral envelope consists of a lipid bilayer where the membrane, envelope and spikestructural proteins are anchored.

Inside the envelope in nucleocapsid, which is formed from multiple copies of nucleocapsid protein whichare bound to positive-sense single-stranded RNA genome in a continuous beads-on-a-string type conformation.

It is called COVID-19 (earlier known as "2019 novel coronavirus").

The official names are:-

Coronavirus disease (COVID-19)

Severe Acute Respiratory Syndrome Coronavirus (SARS-CoV-2)

History of coronavirus:

Coronaviruses are a big family of different viruses. Some of them cause the commoncold in people. Others infect animals, including bats, camels, and cattle.

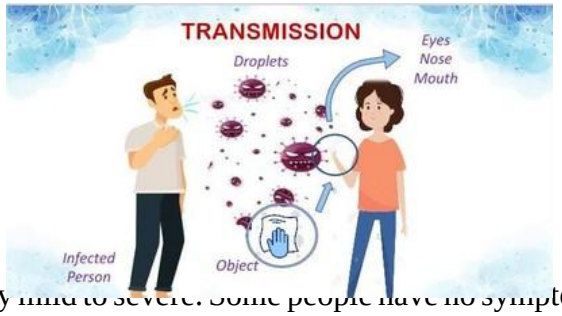
The recent outbreak began in Wuhan, a city in the Hubei province of China. Reports of thefirst COVID-19 cases started in December 2019.

SARS-CoV-2 made the jump to humans at one of Wuhan's open-air "wet markets." They'rewhere customers buy fresh meat and fish, including animals that are killed on the spot.

TRANSMISSION OF CORONAVIRUS:

Coronaviruses mainly spreads from person to person among those in close contact (withinabout 6 feet, or 2 meters). The virus spreads by respiratory droplets released when someone infected with the virus coughs, sneezes, breathes, sings or talks. These droplets can be inhaled or land in the mouth, nose or eyes of a person nearby.

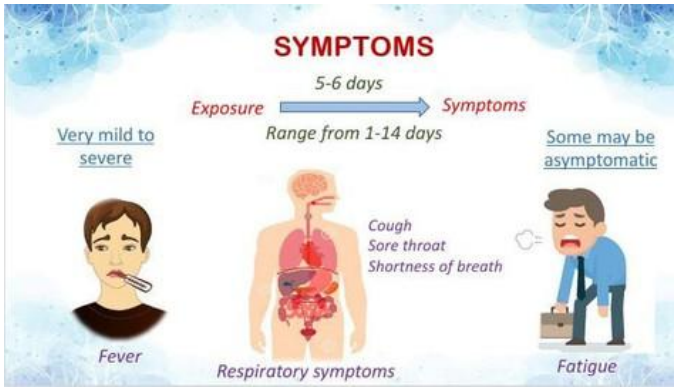
Sometimes the COVID-19 virus can spread when a person is exposed to small droplets thatstay in the air for several minutes or hours — called airborne transmission



SYMPTOMS:

COVID-19 symptoms can be very mild to severe. Some people have no symptoms. Themost common symptoms are fever, cough and tiredness.

Other symptoms may include shortness of breath, muscle aches, chills, sore throat, headache, chest pain, and loss of taste or smell. This list is not all inclusive. Other lesscommon symptoms have also been reported. Symptoms may appear two to 14 days afterexposure.



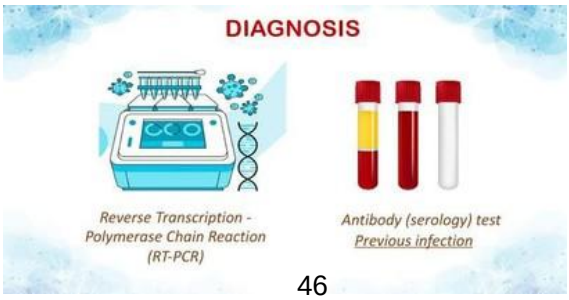
Risk factors:

The risk is higher for anyone in close contact with people who have COVID-19, such as healthcare worker.

Other are.....
Serious heart conditions, such as heart failure, coronary artery disease, orcardiomyopathies
Kidney disease
Chronic obstructive pulmonary disease (COPD)
Obesity, which occurs in people with a body mass index (BMI) of 30 or higherSickle cell disease
A weakened immune system from a solid organ transplantType 2 diabetes

DIAGNOSIS:

PCR test: This tests for the presence of the actual virus's genetic material or its fragments as it breaks down. This is the most reliable and accurate test for detecting active infection. Antibody (serology) test: This tests detects if you've had an immune response (antibodies)to the virus. This means that you've had the virus and your body (immune system, specifically antibodies) has mounted an attack to fight it. The test is detecting those antibodies. This test shouldn't be used to diagnose an active infection.



Treatment:

- Treatment at home:

Rest: It can make you feel better and may speed your recovery.Stay home: Don't go to work, school, or public places.
Drink fluids: Dehydration can make symptoms worse and cause other healthproblems.
Monitor: If your symptoms get worse, call your doctor right away. Don't go to their office without callingfirst.

Ask your doctor about over-the-counter medicines that may help, like acetaminophen to lower your fever.

- Treatment in the Hospital

Check the levels of oxygen in your blood with a clip-on

finger monitor.Listen to your lungs.

Give you a COVID-19 test. This involves putting a 6-inch cotton swab up both sidesof your nose for about 15 seconds.

Give you a chest X-ray or CT scan.

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