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**1st
EDITION**

ORUIKOR GABRIEL JEREMIAH, DrPH

**MANAGEMENT OF
SELECTED DISEASES IN COMMUNITY
HEALTH CARE SETTINGS**

1st edition

By ORUIKOR GABRIEL JEREMIAH, DrPH

Community Medicine Consultant, GF Education & Health Consults

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First Published 2023

Published and printed by: Danchels Nigeria

Location: Zone A, Iba housing estate, Estate main gate, Iba, Lagos.

Tel: 08038054527, **Email:** danchelsnig@gmail.com

ISBN: 978-978-52149-9-4

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DEDICATION

I dedicate this to book to:

The first Love of my life, GOD the creator of all, who gave me this privilege to be a writer and my LOVELY wife, Dr. Mrs Gabriel Jeremiah, who always stood by me in everything good.

ACKNOWLEDGMENTS

I wish to thank some of my Colleagues in west African Union University, International University of Bamenda, My Mentees in TOCHIDWORFI, Nascreative company, the designer of this book, Abundance private schools, Rivers/Bayelsa Union, Jesus witness International Mission, world peace tract ambassadors, The directors of total Child development world International, Dr Paul Nwala, my beloved brother, and my lovely wife, Dr Mrs Gabriel Jeremiah for their support and encouragement during the writing of this book. I am most grateful to Prof DJIBRIL M.Najibou, Alphonsus Izuchukwu Oporum, Pharm. Victor Cheluchukwu Nwankpor, and OKPANACHI MICHAEL, my friends for their useful comments and suggestions which helped to build the manuscript of this book. I am indebted to all my teachers and lecturers, especially Sir ThankGod George Ikor, Sir Samuel, Sir Okiriki Andrew, Dr. Mrs Okocha, and Prof. Wirba Amenu Foven who educated me while I was in School as a student.

I will not fail to recognize my Parents, Mr and Mrs Jeremiah Oruikor Alfred, My Brothers and Sisters, Especially Mr Nsan Jeremiah Oruikor, My Uncles, Capt. George C Iboroma, Mr Oruikor Alfred Alfred and others who had supported me in all my stages of life.

Very importantly, I am grateful to Pst & Dr Mrs Madukor, the founder of Abundance Private Schools, Pst (Engr) & Pst Mrs Tony Chukwu, the Founder of Jesus Witness International Mission, Dr Godwin Onwe, the founder of Christ life Tabernacle, Pst Tonybede Ohaeri, the resident Pastor of House of Victory Bible Church Benin, Dr George Tanko, Director of IUB campus, Pst Useful Nsoghai, Mr & Mrs Tchandjoun, Sis Hortence EWANE, Mr Wisdom Maxwell, Sis Constance Ikpe, Rev Jehoshaphat Omaka, the founder of Good shepherd Internal, Pst. Jacob Amaechi, Rev. Dr Azu Emanuel, the founder of Christ Evangelical team, Mr.Promise Obulor Ahamefula, and Emmanuel UKACHUKWU

Njoku, Chairmen of Rivers/Bayelsa Union Benin, Mr Robert. N.K, the International students affairs IUB, and Mrs Glory Eke, they have been my great encouragers and supporters.

My Profound gratitude goes to Prof. Stephen Nwawolo, the DVC West African Union University, Dr. Bishop Adeyemi Olayemi, the President of West African Union University, Alhaji Onifade A. Abdulkabir, the Registrar of WAUU, Mrs Johnson Alice, the Deputy Registrar WAUU, Dr. Timothee Demahou the Medical director Clinique la Masses Des Figures, Dr Emanuel, the medical director Debrose medical center and Dr. Engr Oladayo. A, the director of heritage foundation, for the opportunity to work in their institutions.

Finally, for all Persons who by one way or the other supported me, I appreciate you.

FOREWORD

This era is filled with different kinds of diseases and many had died, while many are still suffering due to the impacts left by disease. It is a burden on the society as this has and is affecting the world economic. People are now investing and spending all they have in sickness as a recompense of disease invasion.

These diseases are also caused due to our carelessness in the community level. People live and behave anyhow they wish, not minding the factors that poses the risk of being infected by disease.

The public can be as small as a handful of people or as large as a village or an entire city. When there is an increase in the population of people living in an area, the risk of disease is high.

This disease condition historically has become a threat and burden on public health workers who are researching and looking for better ways to prevent, and manage it. In aspect of prevention, it can mean vaccinating children and adults to prevent the spread of disease. Or educating people about the risks of alcohol and tobacco. Public health workers sets safety standards to protect workers and develops school nutrition programs to ensure kids have access healthy food. They work to track disease outbreaks, prevent injuries and shed light on why some of us are more likely to suffer from poor health than others. The many facets employed by public health workers include speaking out for laws that promote smoke-free indoor air and seatbelts, spreading the word about ways to stay healthy and giving science-based solutions to problems.

It is due to this burden that Dr ORUIKOR GABRIEL JEREMIAH, a seasoned Public health Physician placed his hand to promote and make the efforts made by other senior colleagues to have a new shape for easy

and fast understanding of Disease and management at the community level. He wrote this book “*MANAGEMENT OF SELECTED DISEASES IN COMMUNITY HEALTH CARE SETTINGS 1st edition*”, to serve as a manual for managing diseases and preventing it.

His approaches are quick very simple and understanding. He looked at the subject within the following spheres:

Chapter 1, Nature of disease. In this chapter, he defined disease and explained some terms considered associated with disease. Some factors precipitating disease causation was highlighted and within the frame of historical disease impact in the world, some notable diseases of public health important were discussed.

In chapter 2 and 3, he mentioned that some diseases are contagious, which he classified them accordingly and as well, stressed their management.

In chapter 4 to 12, it was a good insight as diseases were classified as air, water, food and sexually associated. In this approach, he looked into the subclasses of these diseases based on the types and groups of organisms that are causative agents. He also discussed those diseases using epidemiology, diagnostic measures and management in different capacities which are not limited to pharmacotherapy, sanitation, vaccination and community engagement.

Finally, in chapter 13-15, He went further to discuss disease as a concern to community health since their lives and economic are threatened by the negative outcomes of disease conditions. He gave different capacities for as a preventive measures to curb or limit the various invasion of disease in the community. The general preventive measures are classified under immunology which he stated involves natural body defense against infections. He did well by also stated the use of vaccines, which he gave details explanation to the underlying approaches. Another option for disease prevention mention by him is

environmental sanitation. Environmental sanitation like he said can play a huge benefits in saving the community from sicknesses.

I must say that it is a rare privilege to assess and review this great work of Dr. ORUIKOR GABRIEL JEREMIAH. He is a writer indeed, for all his books I have reviewed, this one gave me more joy because of the way he presented his work and the perspective of this book, which is channeled to saving lives from the ever increasing sicknesses and poverty. I really commend the author of this book for his courage and contribution in health education and service to humanity.

I therefore, recommend the first edition of this book for students in health sciences and anyone who find it useful to get it, it is very rich and packed with current information on health and life security.

PROF. (DR) NADJIBOU DJIBRIL

-Professor of medicine, University of Parakou,

-Spécialist of cardiovascular emergencies

PREFACE

Historically, the term “Disease” is widely known to many persons as written by many scholars who have taken their researchers in this angle. Disease affect members of the public which include the community. So it’s worthy to address it in the community using community health principles.

A disease causes us discomfort or an uneasy feeling, which may or may not necessarily give a pain sensation. Although “disease-free” and “being healthy” are terms that are often correlated, they are completely different. There is more to “being healthy” than just being “disease-free”. Certain signs and symptoms are used to diagnose a particular disease. And, presence of a particular disease can lead to an unhealthy life.

For people who have taken a career in Health sciences or students in health sciences who would eventually graduate to be health Practitioners, disease management eventually becomes their priority. So it’s crucial and beneficial to understand the science and art of preventing disease, prolonging life and promoting health through the organized efforts and informed choices of society, organizations, public and private, communities and individuals.

This book entitled “ *MANAGEMENT OF SELECTED DISEASES IN COMMUNITY HEALTH CARE SETTINGS 1st edition* “ is well written to enable the readers become familiar with how to promote and protect the health of people and the communities where they live, learn, work and play.

From conducting scientific research to educating about health, people in the field of health work to assure the conditions in which people can be healthy. All the conditions which is needed to get rid of diseases and

maintain good health has been deeply looked into by the author in this book.

Also, the author has strategically placed more emphasis on environmental sanitation, and vaccination as a means of preventing diseases.

This book is not the only book that considered diseases in its perspectives, many authors had written similar topic, but not well understood by the readers due to its volume and complexity of their presentations, so it becomes more tedious to study and obtain a fast understand they need. This singular challenge has made the author to pen down this topic again in a more favourable approach to the readers.

The distinction between this book and others with similar topic is the simplicity of its presentation and deepness of understanding the readers would have when using it. It is presented with not much stories, but the key facts that every reader needs in such a timely book, hence it is pocket friendly.

ORUIKOR GABRIEL JEREMIAH, DrPH

CHAPTER ONE

THE NATURE OF DISEASE

1.0. Overview

A disease or medical condition is an abnormal condition of an organism that impairs bodily functions, associated with specific symptoms and signs. It may be caused by external factors, such as infectious diseases, or it may be caused by internal dysfunctions, such as autoimmune diseases.

In humans, "disease" is often used more broadly to refer to any condition that causes pain, dysfunction, distress, social problems and or death to the person afflicted, or similar problems for those in contact with the person. In its broader sense, it sometimes includes injuries, disabilities, disorders, syndromes, infections, isolated symptoms, deviant behaviours, and atypical variations of structure and function, while in other contexts and for other purposes these may be considered distinguishable categories.

1. 1. DISEASE FACTORS

The following are the factors that precipitate the cause of disease: Environmental, and host.

a. Environmental Factors: these are divided into three namely: biological, physical and social factors.

Biological factors: these includes the infectious agents, reservoirs which may be human, animal or soil, transmitting vectors, plants and animals in their environment which may serve as foods or drugs, antibiotics, antigens and antibodies.

Physical factors: it includes heat, light, air, water, radiation, chemical agent, atmospheric pressure, etc.

Social factor: this is defined as the overall economic and political organisation of a society and the institutions by which individual are integrated into the society at various ages of their lives. Social environment is man- made environment which includes what he has created to make life worth living e.g. housing. It also includes the people's customs, levels of integration of the community, levels and systems of Medicare, the degree of enforcement of health law and code.

b. Host Factors: these are intrinsic factors, genetic (inborn) in a person. It can be classified as:

specific (i.e.) not inborn but acquired by immunisation and natural infection

personality – people working hard, ambitious with strong drive

social class membership: peer grouping, organisation.

1.2. TERMINOLOGIES APPLIED TO DISEASE

Disease causation: This means the thing that makes a disorder or illness happen in people, animals, or plants. Example, bacteria, Virus, fungi, e.t.c

Disease spread: means the act or process by which a particular disease circulates from one another, place to place and country to country. Example HIV spread by blood contact with infected person.

Signs and Symptoms of a Disease:

Signs of a disease are indications of a particular disorder that is observed by a physician but is not apparent to the patient. It can also be said to mean objective manifestations of a disease on a patients.

Symptoms of a disease are indications of a disease by the patient himself. A presenting symptom is one that leads a patient to consult a doctor.

Example, patient hearing imagining voices in a case of schizophrenia.

1.3. Historical impacts of disease

Candidly speaking, it would be difficult to overstate the impact of disease in this world. Since the beginning of human existence on the planet, diseases have played a significant role in the events of every area. Malaria, one of the oldest known diseases, caused drastic deludes in the population of the Greek city- state in the fourth century. The Spanish epidemic of 181- 1919 killed 20 million people around the world than twice the number of people who were killed during World War I. In 1944, a major outbreak of polio around the hickory, North Carolina overwhelmed the area medical facilities, separating sick children from their families and preventing healthy young people from visiting each other for fear of contracting the disease.

Scientific Success About Impact Of Disease

There are scientific achievement/success through the ages and these have greatly alleviated the effect of some of the worst diseases. The formulation of vaccines, the advent of effective mosquitoes control, and the introduction of modern sanitation to urban area have rendered diseases like polio, yellow fever, and cholera all but unheard of in the united states. As a result, average life expectancy in the U.S has risen from 47 years in 1900 to 77 years in 2008.

Unfortunately, through outbreaks of many of the most serious diseases still cause illness and death around the world.

1.4. NOTORIOUS DISEASE IN HISTORY

Malaria- is caused by a parasite transmitted by infected mosquitoes. Symptoms occur ten days to four weeks after being bitten and are similar to symptoms brought on by the flu-chills, fever, sweating, headache, and muscle pain. In serious cases, malaria may cause vomiting anemia (iron deficiency) kidney failure, cornea, and death.

The disease can be prevented by taking anti – malaria drugs and by avoiding mosquito bites in area where malaria infection is common. Those infected with malaria can be treated with prescription drugs, which are most effective when taken early in the course of the disease.

The parasite that most often causes malaria needs warm temperature to grow and thrive, so the disease is typically found in tropical and sub-tropical countries like Nigeria.

Malaria was once common in most of Europe and North Africa, but effective mosquito control and other measures has nearly eradicated the disease in these regions. While only about 1300 cases of malaria are reported in the U.S; each year, 300 to 500 million cases occur around the world- mostly in developing countries like Nigeria and Cameroon – resulting in more than 1 million deaths from malaria globally each year.

Measles:- is a highly contagious disease caused by a virus. Early symptoms include fever, cough, red eyes, and a runny nose. During the first few days, the characteristic measles rash appears, beginning with white spots in the mouth and spreading to a red rash that covers the entire body. The rash typically lasts four to seven days.

Severe cases of measles can cause diarrhea, ear infection, pneumonia, encephalitis (swelling of the brain) and death.

The measles virus is easily spread through airborne droplet expelled by coughing or sneezing and can live in the air for up to two hours after an infected person has been present. After exposure, the virus lives in the body for about two weeks before symptoms appear. There is no specific remedy available for measles, so treatment usually consists of bed rest and easing symptoms.

Measles are still quite common with more than 20 million people infected around the world each year. In the U.S, a widespread utilization campaign has successfully controlled the disease, fewer than 150 cases have been reported since 1997. About half of these cases result from visits to other countries where Measles are still endemic including some developing countries in Europe and Asia.

Mumps: - is a contagious disease that causes painful swelling of the salivary glands. As a result, people infected with mumps sometimes appear to have “chipmunk cheeks”. Other symptoms include fever, headache, sore muscles, and fatigue. Serious complications are rare and may include encephalitis (swelling of the brain), inflammation of the sex organs, and deafness.

The mumps virus transmitted by contact with the respiratory secretions of an infected person like measles, mumps has a relatively long incubation period, with symptoms appearing then two after exposure.

There are no specific treatments available for mumps, but the disease can be prevented by immunization. Following the introduction of the mumps vaccine in 1976, reported mumps cases had declined to fewer than 1000 per year in the U.S. in recent years, however, mumps cases have increased.

Influenza:- More commonly known as “the flu” is caused by a contagious virus. Symptoms include body aches, sore throat, headache, fever, coughing, and chills. Perhaps because influenza is so common, misconceptions about the disease abound. Often, people who experience a bad cold say that have the flu, but this is incorrect. Unlike influenza, colds rarely cause headache, or fever. And despite widespread use of the term “stomach flu”, true influenza does not cause gastrointestinal symptoms.

The flu is spread through air borne respiratory secretions. Symptoms can be serious and the disease can be fatal especially for babies, the elderly, and the people with weakened immune system. When influenza pandemics occur, they spread quickly; often killing large numbers of previously healthy people, from 1918 to 1919, a flu pandemic hit the U.S. in three waves. By the pandemic end, it had spread over the entire globe and killed about 20 million people. Influenza pandemics occurred in 1957 to 1958, and in 1968 to 1969, although they were not as severe as the pandemic of 1918 to 1919.

Influenza is still very common in the U.S. according to the centers for disease control and prevention, between 5 percent and 20 percent of Americans get the flu every year, and about 36,000 cases are fatal. The disease can be treated by antiviral medicines, and the seasonal vaccine is available to prevent against it. Because the virus changes every year, the vaccine must be reformed yearly. Doctors recommend that high-risk members of the population get a vaccine at the beginning of each flu season.

Small pox:- is a serious, contagious, and sometimes fatal infectious disease caused by a virus. There is no specific treatment for small pox disease, and the only prevention is vaccination. The name small pox is derived from the Latin word for “spotted” and refers to the raised bumps that appear on the face and body of an infected person.

Poliomyelitis: (often shortened to ‘polio’) is a viral infection spread by person to person contact.

Symptoms vary according to the type of infection, and three basic patterns are common.

Sub clinical infection, which account for the vast majority of polio cases.

Symptoms may include, fatigue, headache, sore throat, mild fever and vomiting. In some sub clinical infections, no symptoms may appear.

Non- paralytic poliomyelitis. Symptoms may include back pain, neck pain, fever, muscle stiffness, painful rash, and vomiting.

Paralytic poliomyelitis (the most serious kind of polio infection). Symptoms may include fever, breathing difficulty, constipation, headache, muscle pain, muscle spasms, and muscle weakness on one side of the body. Muscle weakness comes on quickly and progresses to paralysis.

Treatments vary according to the form of the disease, and may include antibiotics, pain, relieving medication, and physical therapy to strengthen weak muscles. In 90 percent of cases a complete recovery is possible. Paralytic cases account for the other 10 percent, which are rarely fatal but typically result in permanent disability.

Polio mainly strikes children under the age of 5, and until the 1950s, thousands of children around the world were permanently disabled by polio. In the late 1950s and early 1960s, scientists developed effective vaccines that nearly eradicated polio as a public health problem in industrialized countries. Efforts are underway to eliminate the disease world-wide but around a dozen countries have reported cases in the last two years.

CHAPTER TWO

CONTAGIOUS DISEASES

2.1. Introduction to Contagious Diseases:

A disease that is contagious can be caught by touching people or things that are infected with it.

A contagious (infectious) disease is a clinically evident illness, resulting from the presence of pathogenic microbial agents, including pathogenic viruses, pathogenic bacteria, fungi, protozoa, multicellular parasites, and aberrant proteins known as prions. These pathogens are able to cause disease in animals and or plants. Contagious pathologies are also called communicable diseases or transmissible diseases due to their potential of transmission from one person or species to another by a replicating agent (as opposed to a toxin).

Transmission of an infectious disease may occur through one or more of diverse pathways including physical contact with infected individuals. These infecting agents may also be transmitted through liquids, food, body fluids, contaminated objects, airborne inhalation, or through vector borne spread. Transmissible diseases which occur through contact with an ill person or their secretions, or objects touched by them, are especially infective, and are sometimes referred to as contagious diseases. Infectious (communicable) diseases which usually require a more specialized route of infections, such as vector transmission, blood or needle transmission, or sexual transmission, are usually not regarded as contagious, and thus are not amenable to medical quarantine of victims.

The term infectivity describes the ability of an organism to enter, survive and multiply in the host, while the infectiousness of a disease indicates the comparative ease with which the disease is transmitted to

other hosts. An infection however, is not synonymous with an infectious disease, as an infection may not cause important clinical symptom or impair host function.

2.2 Classification of Contagious Disease:

Among the almost infinite varieties of microorganisms, relatively few cause disease in otherwise healthy individual. Contagious disease results from the interplay between those few pathogens and the defenses of the hosts they infect. The appearance and severity of disease resulting from any pathogen depends upon the ability of that pathogen to damage the host as well as the ability of the host to resist the pathogen. Contagious or infectious microorganisms, or microbes, are therefore classified as either **primary pathogens** or as **opportunistic pathogens** according to the status of host defenses.

2.2.1. Primary Pathogens: Cause disease as a result of their presence or activity within the normal, healthy host, and their intrinsic virulence (*the severity of the disease they cause) is, in part, a necessary consequence of their need to reproduce and spread. Many of the most common primary pathogens of humans only infect humans; however many serious diseases are caused by organisms acquired from the environment or which infect non-human hosts.

2.2.2 Opportunistic pathogens: Organisms which cause contagious/infectious disease in a host with depressed resistance are classified as opportunistic pathogens. Opportunistic disease may be caused by microbes that are ordinarily in contact with the host, such as pathogenic bacteria or fungi in the gastrointestinal or the upper respiratory tract, and they may also result from (otherwise innocuous) microbes acquired from other hosts (as in clostridium difficile colitis) or from the environment as a result of traumatic introduction (as in surgical wound infections or compound fractures). An opportunistic disease which requires impairment of host defenses, which may occur as a result of genetic defects (such as chronic granulomatous disease), exposure to antimicrobial drugs or immunosuppressive chemicals (as might occur following poisoning or cancer chemotherapy), exposure to ionizing radiation, or as a result of

an infectious disease with immunosuppressive activity (such as with measles, malaria or HIV disease). Primary pathogens may also cause more severe disease in a host with depressed resistance than would normally occur in an immunosufficient host.

One way of proving that a given disease is infectious or contagious is to satisfy Koch's postulates (first proposed by Robert Koch), which demands that the infectious agent be identified only in patients and not in healthy controls, and that patients who contract the agent also develop the disease. These postulates were first used in the discovery that mycobacterium species cause tuberculosis. Koch's postulates cannot be met ethically for many human diseases because they require experimental infection of a healthy individual with a pathogen produced as a pure culture. Often, even diseases that are quite clearly

infectious do not meet the infectious criteria. For example, *Treponema pallidum*, the causative spirochete of syphilis, cannot be cultured *in vitro*-however the organism can be cultured in rabbits testes. It is less clear that a pure culture comes from an animal source serving as host than it is when derived from microbes derived from plate culture. Epidemiology is another important tool used to study disease in a population. For contagious or infectious diseases, it helps to determine if a disease outbreak is sporadic (occasional occurrence), endemic (regular cases often occurring in a region), epidemic (an unusually high number of cases in a region), or pandemic (a global epidemic).

2.3 Transmission of Contagious Diseases:

Washing one's hands, a form of hygiene, is the number one way to prevent the spread of contagious or infectious disease.

A contagious disease is transmitted from some sources. Defining the means of transmission plays an important part in understanding the biology of an infectious agent, and in addressing the disease it causes. Transmission may occur through several different mechanisms. Respiratory diseases and meningitis are commonly acquired by contact with aerosolized droplets, spread by sneezing, coughing, talking, kissing or even singing. Gastrointestinal diseases are acquired by

ingesting contaminated food or water. Sexually transmitted diseases are acquired through contact with bodily fluids, generally as a result of sexual activity. Some infectious agents may be spread as a result of contact with a contaminated, inanimate object (known as a fomite), such as a coin passed from one person to another, while other diseases penetrate the skin directly.

Transmission of contagious or infectious disease may also involve a vector. Vectors may be mechanical or biological. A mechanical vector picks up an infectious agent on the outside of its body and transmits it in a passive manner. An example of a mechanical vector is a housefly, which lands on cow dung, contaminating its appendages with bacteria from the faeces, and then lands on food prior to consumption. The pathogen never enters the body of the fly.

Culex mosquitoes (*Culex quinquefasciatus*) are biological vectors that transmit west Nile virus.

In contrast, biological vectors harbor pathogens within their bodies and deliver pathogens within their bodies and deliver pathogens to new hosts in an active manner, usually a bite. Biological vectors are often responsible for serious blood borne diseases, such as malaria, viral encephalitis, Chagas disease, Lyme disease and African sleeping sickness. Biological vectors are usually, though not exclusively, arthropods, such as mosquitoes, ticks, fleas and lice. Vectors are often required in the life cycle of a pathogen. A common strategy used to control vector borne infectious diseases is to interrupt the life cycle of a pathogen by killing the vector.

The relationship between virulence and transmission is complex, and has important consequences for the long term evolution of a pathogen. Since it takes many generations for a microbe and a new host species to co-evolve, an emerging pathogen may hit its earliest victims especially hard. It is usually in the first wave of a new disease that death rates are highest. If a disease is rapidly fatal, the host may die before the microbe can get passed along to another host. However, this cost may be overwhelmed by the short term benefit of higher infectiousness if transmission is linked to virulence, as it is for instance in the case of cholera (the explosive diarrhoea aids the bacterium in finding new host)

or many respiratory infections (sneezing and coughing create infectious aerosols).

2.4 Preventing Transmission of Contagious Disease:

One of the ways to prevent or slow down the transmission of infectious diseases is to recognize the different characteristics of various diseases. Some critical disease characteristics that should be evaluated include virulence distance traveled by victims, and level of contagiousness. The human strains of **Ebola Virus**, for example, incapacitate its victims extremely quickly and kills them soon after. As a result, the victims of this disease do not have the opportunity to travel very far from the initial infection zone. Also, the virus must spread through skin lesions or permeable membranes such as the eye. Thus, the initial stage of Ebola is not very contagious since its victims experience only internal hemorrhaging. As a result of the above features, the spread of Ebola is very rapid and usually stays within a relatively confined geographical area. In contrast, Human Immunodeficiency Virus (HIV) kills its victims very slowly by attacking their immune system. As a result many of its victims transmit the virus to other individuals before even realizing that they are carrying the disease. Also, the relatively low virulence allows its victims to travel long distances, increasing the likelihood of an epidemic.

Another effective way to decrease the transmission rate of infectious disease is to recognize the effects of small world networks. In epidemics, there are often extensive interactions within hubs or groups of infected individuals and other interactions with discrete hubs of susceptible individuals. Despite the low interaction between discrete hubs, the disease can jump to and spread in a susceptible hub via a single or few interactions with an infected hub. Thus, infection rates in small-world networks can be reduced somewhat if interactions between individuals within infected hubs are eliminated. However, infection rates can be drastically reduced if the main focus is on the prevention of transmission jump between hubs. The use of needle exchange programs in areas with a high density of drug users with HIV is an example of the

successful implementation of this treatment method. Another example is the use of ring culling or vaccination of potentially susceptible livestock in adjacent farms to prevent the spread of the foot-and mouth virus in 2001.

General methods to prevent transmission of pathogens may include disinfection and pest control.

CHAPTER THREE

**DIAGNOSIS AND TREATMENT
OF CONTAGIOUS DISEASES**

3.0. Introduction

Diagnosis of contagious disease sometimes involves identifying an infectious agent either directly or indirectly. In practice most minor infectious or contagious diseases such as Warts, cutaneous abscesses, respiratory system infections and diarrhea disease are diagnosed by their clinical presentation. Conclusions about the cause of the disease are based upon the likelihood that a patient came in contact with a particular agent, the presence of a miracle in a community, and other epidemiological considerations. The benefits of identification, however, are often greatly outweighed by the cost, as often there is no specific treatment, the cause is obvious, or the outcome of an infectious is benign.

Specific identification of an infectious agent is usually only determined when such identification can aid in the treatment or prevention of the disease, or to advance knowledge of the course of an illness prior to the development of effective therapeutic or preventative measures. For example, in the early 1980s, prior to the appearance of AZT for the treatment of AIDS the course of the disease was closely followed by monitoring the composition of patient blood sample even though the outcome would not offer the patient any further treatment options. In part, these studies on the appearance of HIV in specific communities permitted the advancement of hypothesis as to the route of transmission of the virus. By understanding how the disease was transmitted, resources could be targeted to the communities at greatest risk in campaigns aimed at reducing the number of new infections. The specific serological diagnostic identification and later genotypic or

molecular identification of HIV also enabled the development of hypothesis as to the temporal and geographical origins of the virus, as well as a myriad of other hypothesis. The developments of molecular diagnostic tools have enabled physicians and researchers to monitor the efficiency of treatment with anti-retroviral drugs. Molecular diagnostics are now commonly used to identify HIV in healthy people long before the onset of illness and have been used to demonstrate the existence of people long before the onset of illness and have been used to demonstrate the existence of people who are genetically resistant to HIV infection. Thus, while there is still no cure for AIDS, there is great therapeutic and predictive benefit to identifying the virus and monitoring the virus levels within the blood of infected individuals, both for the patient and for the community at large.

3.1 Methods of Diagnosis for Contagious Disease:

Diagnosis of contagious disease is nearly always initiated by medical laboratory and physical examination. More detailed identification techniques involve the culture of infectious agent isolated from a patient. Culture allows identification of infectious organisms by examining their microscopic features, by detecting the presence of substances produced by pathogens, and by directly identifying an organism by its genotype. Other techniques (such as x-ray, CAT scans, PET scans or NMR) are used to produce images of internal abnormalities resulting from the growth of an infectious agent. The images are useful in detection of, example, a bone abscess or a spongiform encephalopathy produced by a prion.

3.1.1. Microbial Culture:

It's a principal tool used to diagnose contagious diseases. In a microbial culture, a growth medium is provided for a specific agent. A sample taken from potentially diseased tissue or fluid is then tested for the presence of contagious agent able to grow within that medium. Most pathogenic bacteria are easily grown on nutrient agar, a form of solid medium that supplies carbohydrates and proteins necessary for growth of a bacterium, along with copious amounts of water. A single

bacterium will grow into a visible mound on the surface of the plate called a colony which may be separated from other colonics or melded together into a “lawn”: The size, colour, shape and form of a colony is characteristic of the bacterial species, its specific genetic makeup (its strain), and the environment which supports its growth. Other ingredients are often added to the plate to aid in identification. Plates may contain substances that permit the growth of some bacteria and not others, or that change colour in response to certain bacteria and not others. Bacteriological plates such as these are commonly used in the clinical identification of infectious bacterium. Microbial culture may also be used in the identification of viruses: the medium in this case being cells grown in culture that the virus can infect, and then alter or kill. In the case of viral identification, a region of dead cells results from viral growth, and is called a “plaque”. Eukaryotic parasites may also be grown in culture as a means of identifying a particular agent.

3.1.2. Microscopy:

Another principal tool in the diagnosis of contagious disease is microscopy. Virtually all of the culture techniques discussed above rely, at some point, on microscopic examination for definitive identification of the infectious agent. Microscopy may be carried out with simple instruments, such as the compound light microscope, or with instruments as complex as an electron microscope. Samples obtained from patients may be viewed directly under the light microscope, and can often rapidly lead to identification. Microscopy is often also used in conjunction with biochemical staining technique, and can be made exquisitely specific when used in combination with antibody based techniques. For example, the use of antibodies made artificially fluorescent (fluorescently labeled antibodies) can be directed to bind to and identify a specific antigens present on a pathogen. A fluorescence microscope is then used to detect fluorescently labeled antibodies bound to internalized antigens within clinical samples or cultured cells. This technique is especially useful in the diagnosis of viral diseases, where the light microscope is incapable of identifying a virus directly.

3.1.3 Biochemical Tests:

Biochemical tests used in the identification of infectious agent include the detection of metabolic or enzymatic products of metabolic or enzymatic products characteristic of a particular infectious agent since bacteria ferment carbohydrates in patterns characteristic of their genus and species, the detection of fermentation products is commonly used in bacterial identification. Acids, alcohols and gases are usually detected in these tests when bacteria are grown in selective liquid or solid media.

3.1.4 Molecular Diagnosis:

Technologies based upon the polymerase chain reaction (PCR) method will become nearly ubiquitous gold standards diagnostics of the near future, for several reasons. **First**, the catalog of infectious agents has grown to the point that virtually all of the significant infectious agents of the human population have been identified. **Second**, an infectious agent must grow within the human body to cause disease; essentially it must amplify its own nucleic acids in order to cause a disease. This amplification of nucleic acid in infected tissue offers an opportunity to detect the infectious agent by using PCR. **Third**, the essential tools for directing PCR, primers, are derived from genomes of infectious agents, and with time those genomes will be known, if they are not already.

CHAPTER FOUR

AIRBORNE DISEASE:

4.0 Introduction

An Airborne disease is an infectious disease acquired by inhalation of infective agents. Infective agents include viruses, bacteria, fungi and rickettsia. Reservoir is mainly human, and carriers play a major role in their epidemiology. The Mode of transmission by droplets, droplet nuclei and dust. Host immunity plays a role in epidemiology.

4.1 Impact of Airborne Disease

In other vector-borne diseases, flies and other insects transmit the infectious parasite. Sleeping sickness (African trypanosomiasis) for instance, is transmitted by the bite of the tsetse fly. Leishmaniasis is transmitted by the sandfly, again through a parasite-contaminated bite. This disease is a major problem in parts of Africa, Latin America, and the Middle East, where some 2 million cases occur each year. In its deadliest form, visceral leishmaniasis, fatality rates can reach 100 percent if untreated. In its more common form, the disease produces painful ulcers on the face, arms, and legs. WHO estimates that some 350 million people are at risk of contracting the leishmaniasis. As with other vector-borne diseases, its spread is accelerated by development projects such as road building or forest exploitation, which bring people into contact with the disease vector. Leishmaniasis is reappearing in some areas where it had once been controlled, in part because of the cessation of insecticide spraying to control malaria. A beneficial side effect of DDT, it turns out, was that it kept sandflies in check.

Chagas disease, also known as American trypanosomiasis, is unique to the Americas, where housing conditions pose the biggest risk factor. In this case the culprit, the parasite *T. cruzi*, is carried by both wild and domesticated animals. It is usually transmitted, however, by a blood-

sucking bug that lives in thatched roofs. About one third of those infected develop a chronic form of the disease, which can lead to heart damage and death. WHO estimates that Chagas disease is the leading cause of cardiac death among young adults in parts of South America.

Airborne disease is commonly known as ARI, acute respiratory infections kill more than 4 million people per year and are the leading cause of death among children under age 5. This range of infections, which includes pneumonia in its most serious form, accounts for more than 8 percent of the global burden of disease. ARI's reach is global: it is the most frequent disease worldwide and a common causes of visits to paediatricians in the industrialized countries, although essentially all deaths from ARI occur in the developing world.

The risk factors for ARI are numerous and difficult to sort out. Caused by different viruses or bacteria, ARI is closely associated with poverty. Overcrowding and unsanitary household conditions favour the transmission of the disease, which is spread by droplets from a cough or a sneeze or unwashed hands. Death most often strikes those children who are already weakened by low birth weight, other infections, and malnutrition.

Several other factors seem to exacerbate the disease. Exposure to tobacco smoke increases the risk of contracting these infections, and many studies implicate both indoor and outdoor air pollution. Indoor air pollution has been the focus of particular concern, specifically, the soot and smoke associated with the burning of biomass fuels such as wood, coal, or dung. Many people in the developing world, mostly in rural areas, rely on biomass fuels for heating or cooking. A cause-and-effect relationship between indoor air pollution and ARI has been difficult to prove, however, in part because people who use biomass fuels tend to be poor and exposed to multiple risks such as overcrowding, tobacco smoke, and malnutrition. Even so, the World Bank estimated in 1992 that switching to better fuels could halve the number of pneumonia deaths.

Other airborne diseases also thrive in conditions of poverty, exploiting enclosed spaces, crowding, and poor hygienic conditions. Tuberculosis (TB), to name just one, killed an estimated 3 million people in 1996,

and nearly 7.5 million others developed the disease. TB is the single largest cause of adult death from infectious diseases. Roughly 95 percent of all TB sufferers are in the developing world, mostly in Southeast Asia, Western Pacific, and Africa – many in the slums of poor cities. In recent years, however, TB has resurfaced in developed countries, where it is concentrated among poor populations.

Measles and diphtheria, also diseases of crowding and poverty, have been all but eliminated in the developed world since the advent of successful vaccines. In the developing world, however, measles still affects 42 million children per year who lack access to the vaccine; roughly 1 million of these children die. Since 1990, diphtheria has resurfaced in the former Soviet Union, triggered by social disruption and a drop in immunization rates.

Measles and diphtheria are just two of a cluster known as childhood (or vaccine-preventable) diseases. Other familiar diseases in this group are neonatal tetanus, poliomyelitis, and pertussis. This cluster, all linked with environmental conditions, accounts for nearly 15 percent of the total disease burden globally for children under age 5. Despite widespread immunization programs, these diseases nonetheless claimed the lives of 1,985,000 children in 1990.

4.2 Control of Airborne disease

1. At level of infective agent, mode of transmission and susceptible host
2. At level of mode of transmission includes good ventilation and personal hygiene.
3. At level of host includes specific immunization and chemoprophylaxis.

CHAPTER FIVE

COMMON AIRBORNE DISEASES

5.0 Definition of Airborne Diseases:

Airborne diseases are those diseases which are caused by pathogenic microbial agents which get discharged through coughing, sneezing, laughing, or through close personal contact. These pathogens ride on either dust particles or small respiratory droplets and can stay suspended in air and or are capable of travelling distances on air currents.

Many common infections can spread by airborne transmission at least in some cases, including; anthrax (inhalational), chickenpox, influenza, smallpox, and Tuberculosis.

5.1 An overview of some Airborne diseases:

Diphtheria: Respiratory diphtheria causes a sore throat and fever, and sometimes swelling of the neck. In severe cases it can cause a membrane to form over the throat, which results in breathing problems. Cutaneous diphtheria affects the skin, causing infected lesions to form. Diphtheria can lead to coma and death if it goes untreated.

An infected person usually spreads the disease by coughing or sneezing. The person expels droplets containing the diphtheria bacteria, which are then inhaled by another person. The disease is treated by hospitalization and antibiotics.

Diphtheria was once very common in the U.S., with hundreds of thousands of cases occurring every year. Since the introduction of a vaccine in the 1920s, cases of diphtheria in the U.S. have declined greatly, with less than one case reported each year since 2000. But while mandatory vaccines for school children have gone a long way toward controlling diphtheria in the U.S., the disease is still endemic in many developing countries.

Measles: Is a highly contagious disease caused by a virus. Early symptoms include fever, cough, red eyes, and a runny nose. During the first few days, the characteristic measles rash appears, beginning with white spots in the mouth and spreading to a red rash that covers the entire body. The rash typically lasts four to seven days severe cases of measles can cause diarrhea, ear infection, pneumonia, encephalitis (swelling of the brain), and death.

The measles virus is easily spread through airborne droplets expelled by coughing or sneezing and can live in the air for up to two hours after an infected person has been present. After exposure, the virus lives in the body for about two weeks before symptoms appear. There is no specific remedy available for measles, so treatment usually consists of bed rest and easing symptoms.

Measles are still quite common, with more than 20 million people infected around the world each year. In the U.S., a widespread immunization campaign has successfully controlled the disease, and fewer than 150 cases have been reported since 1997. About half of these cases result from visits to other countries where measles is still endemic-including some developed countries in Europe and Asia.

Mumps: Is a contagious viral disease that causes painful swelling of the salivary glands. As a result, people infected with mumps sometimes appear to have “chipmunk cheeks”. Other symptoms include fever, headache, sore muscles, and fatigue. Serious complications are rare, and may include encephalitis (swelling of the brain), inflammation of the sex organs, and deafness.

The mumps virus is transmitted by contact with the respiratory secretions of an infected person. Like measles, mumps has a relatively long incubation period, with symptoms appearing more than two weeks after exposure. There are no specific treatments available for mumps, but the disease can be prevented by immunization. Following the introduction of the mumps vaccine in 1967, reported mumps cases had declined to fewer than 1000 per year in the U.S. In recent years, however, mumps cases have increase.

Influenza: More commonly known as “the flu”, is caused by a contagious virus. Symptoms include body aches, sore throat, headache, fever, coughing, and chills. Perhaps because influenza is so common, misconceptions about the disease abound. Often, people who experience a bad cold say that have the flu, but this is incorrect. Unlike influenza, colds rarely cause headaches or fever. And despite widespread use of the term “stomach flu” true influenza does not cause gastrointestinal symptoms.

The flu is spread through airborne respiratory secretions. Symptoms can be serious, and the disease can be fatal-especially for babies, the elderly, and people with weakened immune systems. When influenza pandemics occur, they spread quickly, often killing large numbers of previously health people. From 1918 to 1919, a flu pandemic hit the U.S. in three waves. By the pandemics end, it had spread over the entire globe and killed about 20 million people. Influenza pandemics occurred in 1957 – 1958, and in 1968 – 1969, although they were no where near as severe as the pandemic of 1918 – 1919.

Influenza is still very common in the U.S. According to the centers for Disease control and prevention, between 5 percent and 20 percent of Americans get the flu every year, and about 36,000 cases are fatal. The disease can be treated by antiviral medicines, and a seasonal vaccine is available to protect against it. Because the virus changes every year, the vaccine must be reformulated yearly. Doctors recommend that high-risk members of the population get a vaccine at the beginning of each flu season.

Smallpox: Is a serious, contagious and sometimes fatal infectious disease caused by a virus? There is no specific treatment for smallpox disease, and the only prevention is vaccination. The name smallpox is derived from the Latin word for “spotted” and refers to the raised bumps that appear on the face and body of an infected person.

Yellow Fever: The disease typically occurs in tropical areas, particularly in parts of South America and sub-Saharan Africa. Before its cause was known, yellow fever was also common in the U.S. and

outbreaks killed thousands in port cities, include Philadelphia, New York, Baltimore, Norfolk, and New Orleans. In 1900, U.S. Army physicians made the connection between yellow fever and mosquito bites, and subsequent efforts to control mosquitoes led to a significant decline in yellow fever cases in the U.S. The last epidemic of yellow fever occurred in New Orleans in 1905.

Yellow fever symptoms usually appear three to six days after being bitten by an infected mosquito, and they develop in three stages. In the **first stage**, symptoms include fever, headache, muscle aches, vomiting and jaundice. This lasts for three to four days before the **second stage** remission-beings. During remission, the fever and other symptoms go away. Most people recover at this stage, but some more on to the **third stage**-intoxication. This stage is the most serious and involves liver and kidney failure, bleeding disorders, delirium, coma, and secures. Yellow fever that reaches this third stage is often fatal. Although individual symptoms can be treated, no effective treatments exist for the disease itself. A vaccine that effectively prevents yellow fever has been widely available since the 1950s.

5.2 Examples of some Airborne Diseases: (Etiology, Symptoms, prevention and treatment)

5.2.1. Meningitis:

Is an inflammation of the meninges due to infection by virus or bacteria. Meningitis causes an intense headache, fever, loss of appetite, intolerance to light and sound, rigidity of muscles, especially those in the neck or knee. Example, kernig's sign (a

symptom of meningitis in which the hamstring muscles/tendons at the back of the knee, becomes so stiff, such that the patient is unable to extend his legs at the knee when the thighs are held at a right angle to the body). In severe cases, meningitis results to cases of convulsions, vomiting and delirium leading to death.

The most important causes of bacterial meningitis in young children are Haemophilus influenza and Neisseria meningitides is against Haemophilus meningitis is now routine (HIB VACCINE).

5.2.2. HIB VACCINE:

Is a vaccine that gives protection against the bacterium *Haemophilus influenzae* type B (Hib). Before the introduction of the vaccine in the UK in 1992, Hib was the commonest cause of meningitis in children under the age of two. The vaccine, which has an excellent safety record, is currently given with the triple DPT Vaccine at two, three and four months of age.

In **meningococcal meningitis** (also known as cerebrospinal fever and spotted fever), the symptoms appear suddenly and the bacteria can cause a widespread meningococcal infection culminating in meningococcal septicemia, with its characteristic hemorrhagic rash anywhere on the body. Unless rapidly diagnosed and treated, death can occur within a week.

Bacterial meningitis is treated with large dose of antibiotics administered as soon as possible, after diagnosis. With the exception of herpes simplex encephalitis (which is treated with acyclovir) viral meningitis does not respond to drugs but normally has a relatively benign prognosis.

5.2.3 Measles:

Is a highly infectious virus disease that tends to appear in epidemics every 2-3 years and mainly affects children. After an incubation period of 8 – 15 days, symptoms resembling those of a cold develop accompanied by a high fever. Small red spots with white centers (Koplik's spots) may appear on the inside of the cheeks. On the third to fifth day a blotchy slightly elevated pink rash develops, first behind the ears, then on the face and elsewhere. It lasts 3-5 days. The patient is infectious throughout this period. In most cases the symptoms soon subside but patients are susceptible to pneumonia and middle ear infections. Complete recovery may take 2-4 weeks. Severe complications include encephalitis (one in 1000 cases) and subacute sclerosing panencephalitis. Measles is a common cause of childhood mortality in malnourished children, particularly in the developing world. Vaccination against measles provides effective immunity (MMR VACCINE).

5.2.4. MMR VACCINE:

Is a combined vaccine against measles, mumps, and German measles (Rubella). It is currently recommended that this vaccine is given to all children between 13 and 15 months old with a booster dose at 3.5-4 years. Specific contraindications include immunosuppression and allergy to eggs.

5.2.5. Histoplasmosis:

Histoplasmosis is an infection caused by inhaling spores of the fungus *Histoplasma capsulatum*. The primary pulmonary form usually produces no symptoms or harmful effects and is recognized retrospectively by x-rays and positive histoplasmin skin testing. Occasionally, progressive Histoplasmosis, which resembles tuberculosis, develops. The fungus may spread via the bloodstream to attack other organs, such as the liver, spleen, lymph nodes, or intestine. Symptomatic disease is treated with intravenous amphotericin – B. The spores are found in soil contaminated by feces, especially from chickens and bats. The disease is endemic in the northern and central USA, Argentina, Brazil, Venezuela, and parts of Africa.

Chickenpox:

Chickenpox is an airborne and a highly infectious disease caused by a herpes virus (the varicella-zoster virus) that is transmitted by airborne droplets. After an incubation period of 11-18 days, a mild fever develops, followed after about 24 hours by an itchy rash of red pimples that soon change to vesicles. These usually start on the trunk or scalp and spread to the face and limbs, the crust over and resolve after about 12 days.

Treatment is aimed at reducing the fever and controlling the itching (e.g. by the application of calamine lotion), scarring is unusual.

Complications are rare but include secondary infection and occasionally encephalitis. The patient is infectious from the onset of symptoms until all the spots have gone. One attack usually confers life-long immunity, although the virus may reactivate at a later date and cause shingles. In adult patients who are particularly venerable, e.g. those with AIDS or

who are otherwise immuno-suppressed, chickenpox can be a serious disease, which may be treated with acyclovir or famciclovir (varicella).

5.2.7. Tuberculosis:

Tuberculosis is airborne and an infectious disease caused by the bacillus mycobacterium tuberculosis (first identified by Koch in 1882) and characterized by the formation of nodular lesions (tubercles) in the tissues.

In **pulmonary tuberculosis** – formally known as consumption and phthisis (wasting) – the bacillus is inhaled into the lungs where it sets up a primary tubercle and spreads to the nearest lymph nodes (the primary complex). Natural immune defenses may heal it at this stage, alternatively the disease may smolder for months or years and fluctuate with the patient's resistance. Many people become infected but show no symptoms. Others develop a chronic infection and can transmit the bacillus by coughing and sneezing. Symptoms of the active disease include fever, night sweats, weight loss, and the spitting of blood.

In some cases the bacilli spread from the lungs to the blood stream setting up millions of tiny tubercles throughout the body (miliary tuberculosis), or migrate to the meninges to cause tuberculous meningitis. Bacilli entering by the mouth, usually infected cow's milk, set up a primary complex in abdominal lymph nodes, leading to peritonitis, and sometimes spread to other organs, joints, and bones (POT Disease).

Tuberculosis is curable by various combinations of the antibiotics streptomycin, ethambutol, isoniazid (INH), rifampicin, and pyrazinamide.

Preventive measures in the UK include the detection of cases by x-ray screening of vulnerable populations and inoculation with BCG vaccine of those with no immunity to the disease (the tuberculin test identifies which people require vaccination).

5.2.8. Influenza:

Influenza is an airborne and a highly contagious virus infection that affects the respiratory system. The viruses are transmitted by coughing and sneezing.

Symptoms commence after an incubation period of 1-4 days and include headache, fever, loss of appetite, weakness, and general aches and pains. They may continue for about a week. With bed rest and aspirin most patients recover, but a few may go on to develop pneumonia, either a primary influenza viral pneumonia or a secondary bacterial pneumonia. Either of these may lead to death from hemorrhage within the lungs. The main bacterial organisms responsible for secondary infection are streptococcus pneumonia, Haemophilus influenza, and staphylococcus aureus, against which appropriate antibiotic therapy must be given. An influenza infection provides later protection only against the specific strain of virus concerned, the same holds true for immunization.

CHAPTER SIX

EPIDEMIOLOGY OF SOME AIRBORNE DISEASES

6.1 Measles:

Epidemiologically, Measles is an acute infectious disease characterized by fever, cough, and characteristic skin rash.

Its occurrence is worldwide.

Reservoir of measles infection is man, and transmission is by droplets, contact and fomites.

Incubation period is usually 10 days.

Symptoms and signs include Koplik's spots and skin rash.

Measles is more severe in malnourished children.

6.1.1 Diagnosis and control.

Diagnosis of measles is clinical and laboratory.

Control of measles is by active immunization.

6.1.2 Meningococcal Meningitis

Epidemiologically, meningococcal meningitis is an acute infectious disease caused by *Neisseria meningitis* – A, B, C, W-135 and Y.

Its occurrence is worldwide, epidemics occur in meningitis belt in Africa: 5 – 15 degrees N of equator.

Humans are reservoir of infection, and carriers play a major role.

Mode of transmission is by airborne droplets and direct contact.

Risk of transmission is increased by travel, migration, pilgrimages and overcrowding.

In countries in meningitis belt, peak incidence is in age group 5 – 10 years.

6.1.3 Diagnosis and control

1. Bacteriological examination of nasopharyngeal swabs, blood and cerebrospinal fluid
2. CSF shows cloudy fluid, pus cells, raised protein content and low or absent glucose.

Control

1. Environmental control
2. Management of patients with rifampin
3. Immunization
4. Surveillance.

A rate of 15 cases per 100,000 per week averaged over two consecutive weeks is indicative of an epidemic.

6.2 Histoplasmosis:

Epidemiologically, Histoplasmosis is an infection with causative agent known as *Histoplasma capsulatum*.

It is endemic in Africa, South America and Far East.

Reservoir is in soil-bat caves, areas with pigeon droppings.

Mode of transmission is by inhalation of spores.

Incubation period is 1-2 weeks.

Symptoms – acute respiratory illness which is usually self-limiting.

Disseminated lesions may occur.

6.2.1 Diagnosis and Control.

1. Isolation on culture of sputum or biopsy material-culture media is Sabouraud's medium
2. Serological test.

Control

1. avoiding exposure
2. treatment with Amphotericin B.

CHAPTER SEVEN

EPIDEMIOLOGY OF TUBERCULOSIS:

7.0 Tuberculosis:

Epidemiologically, tuberculosis is a chronic infection caused by the bacteria *Mycobacterium tuberculosis* (and occasionally other variants of mycobacterium). It usually involves the lungs, but other organs of the body can also be involved.

7.1 An overview of Tuberculosis:

Today, tuberculosis (TB) tends to be concentrated among inner city dwellers, ethnic minorities and recent immigrants from areas of the world where the disease is still common. Alcoholics, who are often malnourished, are at high risk of developing the disease, as are people infected with HIV. It can occur anywhere, and no one is exempted from the threat of infection.

TB is caused by a germ that is transmitted from person to person by airborne droplets. Usually this infections passed on as a result of very close contact, so family members of an infected person are endangered if the person continues to core in the same household and has not undergone proper treatment (The family should take the precaution of seeing a doctor and getting a skin test).

If an individual with active TB coughs or sneezes without covering the mouth and nose, droplets containing the tuberculosis germs are sprayed into the air and may be inhaled by anyone near the person. A tissue should always be used to cover the nose and mouth when coughing, sneezing or spitting, and hands should be washed promptly.

The vast majority of people who have TB germs in their bodies do not have an active case of the disease. Even if the disease is active, the

disease is quite advanced. TB in children often occurs with childhood diseases. A simple skin test is available to detect individuals who have been or are infected with TB germ. Those who have been infected will have a reaction where the skin becomes swollen. Once infected, most persons will generally test positive for the rest of their lives.

A positive reaction to the tuberculin test does not mean the person is ill or contagious to others. It means that the germs causing tuberculosis have been or are present in the body, and unless other symptoms are evident, the germs are probably not active. Their doctor may want to treat them to eliminate the germs so that a more serious case of active TB can be prevented.

7.2 Etiology of Tuberculosis:

Tuberculosis of the lungs usually results in no or minimal symptoms in its early stages. In most persons the primary infection is contained by the body's immune system, and the lesion, called a tubercle, becomes calcified. In many, the infection is permanently arrested. In others the disease may break out again and become active years later, usually when the body's immune defenses are low. Untreated, the infection can progress until large areas of the lung and other organs are destroyed.

7.3 Symptoms of Tuberculosis:

Only about 10 percent of those infected with TB develop the disease. The first symptoms of an active case of TB may be so common place that they are often dismissed as the effects of a cold or flu. The individual may get tired easily, feel slightly feverish or cough frequently. It usually goes away by itself, but in about half the cases, it will return.

For people who have the disease, TB can cause lung or pleural (the lining of the lung) disease or it may spread through the body via the blood. Often people do not seek the advice of a doctor until they have pronounced symptoms, such as pleurisy (a sharp pain in the chest when breathing deeply or coughing) or the spitting up of blood. Neither of these symptoms is solely of tuberculosis but they should not be ignored.

Other symptoms include fever, loss of appetite, weight loss and night sweats.

About 15 percent of people with the disease develop TB in an organ other than the lung, such as the lymph nodes, Gastro-intestinal tract, and bones and joints.

7.4 **Epidemiology of Tuberculosis:**

The incidence of tuberculosis of the lungs, the “white plague” that formerly affected millions of people, declined from the 1950s until 1984. sanatoriums were closed and routine screening was abandoned in the United States. Then, between 1984 and 1992, the incidence increased by 20% chiefly because of immigration from countries where it is common and because of AIDS.

Acquired Immunodeficiency Syndrome (AIDS) is a fatal disease caused by a rapidly mutating retrovirus that attacks the immune system and leaves the victim vulnerable to infections, malignancies, and neurological disorders.

Renewed efforts at control and advances in treatment of tuberculosis have been rewarded with incidence declines each year, amounting to a total decline of 31% from 1992 to 1998.

Worldwide the outlook has been far less encouraging. In 1993, the World Health Organization (WHO) declared TB a global health emergency. Approximately one third of the world’s population is infected, and an estimated 1.6 million die each year. The vast majority of new cases occur in sub-Saharan Africa. Spread of TB is especially rapid in areas with poor public health services and crowded often go together. Areas where living condition are disrupted by wars, families, and natural disaster also is heavily affected.

Especially alarming has been the spread of drug-resistant strains of TB. By the late 1990s scientific experts and international health officials warned that drug-resistant strains were spreading faster than had been anticipated. Bacteria can survive and become drug resistant in patients whose treatment is not properly monitored and seen to completion. Multidrug resistant TB strains are resistant to two or more of the commonly prescribed first-line drugs, while extreme drug resistant strains are also resistant to three or classes of the more toxic second line

drugs. Some believe that unless major new treatment strategies are initiated in source countries, drug-resistant TB will eventually become epidemic even in areas with good control programs such as Europe and America.

7.5 Diagnosis of Tuberculosis:

If a person has a significant reaction upon being tuberculin skin-tested for the first time additional laboratory and x-ray examinations are necessary to determine if the individual has active TB.

Tuberculosis can mimic other diseases, such as pneumonia, lung abscesses, tumors and fungal infections, or occur along with them. For a proper diagnosis, therefore, a doctor will rely on symptoms and other physical signs; a person's history of exposure to TB and x-rays that may show evidence of TB infection (usually in the form of lesions or cavities in the lungs). TB bacilli grown in cultures of sputum or other specimens provide a positive diagnosis.

7.6 Treatment of Tuberculosis:

With treatment, the chance of full recovery is good. Although several treatment protocols for active TB are in wide use by specialists, and protocols sometimes change due to advance in our understanding of optimal therapy, they generally share three principles:

- a. The regimen must include several drugs to which the organisms are susceptible.
- b. The patient must take the medication on a regular basis.
- c. Therapy must continue for a sufficient time.

Also, treatment recommendations are subject to change depending on both the characteristics of the particular organism being treated and newer advances in therapeutic agents. Thus, consultation on treatment strategies with local public health and infectious disease experts is always advisable.

Isoniazid (INH) is one of the most common drugs used for TB. Inexpensive, effective and easy to take, it can prevent most cases of TB and when used in conjunction with other drugs, cure most TB. INH preventive treatment is recommended for individuals who have:

- i. Close contact with a person with infectious TB
- ii. Positive tuberculin skin test reaction and an abnormal chest x-ray that suggests inactive TB.
- iii. A tuberculin skin test that converted from negative to positive within the past two years.
- iv. A positive skin test reaction and a special medical condition (for example, AIDS or HIV infection or diabetes) or who are on corticosteroid therapy.
- v. A positive skin test reaction, even with none of the above risk factors (in those under 35).

Isoniazid and **rifampin** are the keystones of treatment, but because of increasing resistance to them, pyrazinamide and either streptomycin sulfate or ethambutol HCL is added to regimens. If the patient is unable to take pyrazinamide, a nine-month regimen of Isoniazid and rifampin is recommended.

Even if susceptibility testing reveals that the patient is infected with an Isoniazid-resistant and ensuring a good outcome. Six months is the minimum acceptable duration of treatment for all adults and children with culture-positive TB.

Drug resistance may be either primary or acquired. Primary resistance occurs in patients who have had no previous antimycobacterial treatment. Acquired resistance occurs in patients who have been treated in the past, and it is usually as a result of non-adherence to the recommended regimen or incorrect prescribing.

It has been estimated that one in seven cases of tuberculosis is resistant to drugs that previously is resistant to drugs that previously cured the disease. Resistance arises when patients fail to complete their drug therapy, lasting six months or longer. The hardest TB bacteria are allowed to survive as a result, and as they multiply, they spread their genes to a new generation of bacteria and to new victims.

The drug-resistant forms of TB that do not respond to the usual drug therapy might be treatable by other, sometimes more toxic drugs. Officials of the center for Disease control and prevention call for aggressive intervention to prevent the further spread of drug-resistant TB, including finding “every TB patient” and ensuring that patients

complete their drug therapy. To accomplish this, increasing use of directly observed therapy (DOT) is being used—that is, the actual, documented observation of the patient when he or she takes the medicine. This method has been shown to reduce the likelihood of treatment failures.

Overall, it is critical to consult with a physician about the optimal course of therapy for any given case of tuberculosis. In turn, your physician will likely consult with local public health experts to determine if any local circumstances (such as drug-resistant TB) apply to a particular case.

CHAPTER EIGHT

EPIDEMIOLOGY OF CHICKENPOX AS AN AIRBORNE DISEASE

8.0 Chickenpox:

Alternative name for chickenpox is varicella

8.1 Description of Chickenpox:

Chickenpox is one of the classic childhood diseases. A child or adult with chickenpox may develop hundreds of itchy, fluid-filled blisters that burst and form crusts. Chickenpox is caused by a virus.

8.2 Etiology of Chickenpox:

The virus that causes chickenpox is varicella-zoster, a member of the herpes virus family. The same virus also causes herpes zoster (shingles) in adults.

In a typical scenario, a young child is covered in pox and out of school for a week. The first half of the week the child feels miserable from intense itching; the second half from boredom. Since the introduction of the chickenpox vaccine, classic chickenpox is much less common.

Chickenpox can be spread very easily to others. You may get chickenpox from touching the fluids from a chickenpox blister, or if someone with chickenpox coughs or sneezes near you. The vaccine usually prevents the chickenpox disease completely or makes the illness very mild. Even those with mild illness may be contagious.

When someone becomes infected, the pox usually appears 10 – 21 days later. People become contagious 1 to 2 days before breaking out with pox. They remain contagious while uncrushed blisters are present.

Most cases of chickenpox occur in children younger than 10. The disease is usually mild, although serious complications sometimes occur. Adults and older children usually get sicker than younger children do.

Children whose mothers have had chickenpox or have received the chickenpox vaccine are not very likely to catch it before they are 1 year old. If they do catch chickenpox, they often have mild cases. This is because antibodies from their mother's blood help protect them. Children under 1 year old whose mothers have not had chickenpox or the vaccine can get severe chickenpox.

Severe chickenpox symptoms are more common in children whose immune system does not work well. This may be caused by an illness or medicines such as chemotherapy and steroids.

8.3 Symptoms of Chickenpox:

Most children with chickenpox act sick, with symptoms such as a fever, headache, tummy ache, or loss of appetite for a day or two before breaking out in the classic pox rash. These symptoms last 2 to 4 days after breaking out.

The average child develops 250 to 500 small, itchy, fluid-filled blisters over red spots on the skin.

1. The blisters often appear first on the face, trunk, or scalp and spread from there. Appearance of the small blisters on the scalp usually confirms the diagnosis.
2. After a day or two, the blisters become cloudy and then scab. Meanwhile, new crops of blisters spring up in groups. The pox often appears in the mouth, in the vagina, and on the eyelids.
3. Children with skin problems such as eczema may get more than 1500 pox.

Most pox will not leave scars unless they become infected with bacteria from scratching.

Some children who have had the vaccine will still develop a mild case of chickenpox. They usually recover much more quickly and have only

a few pox (less than 30). These cases are often hardened to diagnose. However, these children can still spread chickenpox to others.

8.4 Clinical Examination and Tests of Chickenpox:

Chickenpox is usually diagnosed from the classic rash and the child's medical history. Blood tests, and tests of the pox blisters themselves, can confirm the diagnosis if there is any question.

8.5 Treatment of Chickenpox:

In most cases, it is enough to keep children comfortable while their own bodies fight the illness. Oatmeal baths in lukewarm water provide a crust, comforting coating on the skin. An oral antihistamine can help to ease the itching, as can topical lotions. Trim the finger nails short to reduce secondary infections and scarring.

Safe antiviral medicines have been developed. To work well, they usually must be started within the first 24 hours of the rash.

1. For most otherwise healthy children without severe symptoms, antiviral medications are usually not used. Adults and teens, which are at risk for more severe symptoms, may benefit if the case is seen early in its course.
2. For those with skin conditions (such as eczema or recent sunburn), lung condition (such as asthma), or those who have recently taken steroids, the antiviral medicines may be very important. The same is also true for adolescents and children who must take aspirin on an ongoing basis.
3. Some doctors also give antiviral medicines to people in the same household who subsequently come down with chickenpox. Because of their increased exposure, they would normally experience a more severe case of chickenpox.

Do not give Aspirin to someone who may have chickenpox. Use of aspirin has been associated with a serious condition called Reyes syndrome. Ibuprofen has been associated with more severe secondary infections. Acetaminophen may be used.

Until all chickenpox sores have crusted over or dried out, avoid playing with other children, going back to school, or returning to work.

8.6 An overview of Chickenpox:

The outcome is generally excellent in uncomplicated cases. Encephalitis, pneumonia, and other invasive bacterial infections are serious, but rare, complications of chickenpox.

Once you have had chickenpox, the virus usually remains dormant or asleep in your body for your lifetime. About 1 in 10 adults will experience shingles when the virus re-emerges during a period of stress.

8.7 Some complications of Chickenpox:

- a) Women who get chickenpox during pregnancy are at risk for congenital infection of the fetus.
- b) Newborns are at risk for severe infection, if they are exposed and their mothers are not immune.
- c) A secondary infection of the blisters may occur.
- d) Encephalitis is a serious, but rare complication.
- e) Reye's syndrome, pneumonia, myocarditis and transient arthritis are other possible complications of chickenpox.
- f) Cerebella ataxia may appear during the recovery phase or later. This is characterized by a very unsteady walk.

8.7.1 Medical check-up:

Call your health care provider if you think that your child has chickenpox or if your child is over 12 months of age and has not been vaccinated against chickenpox.

8.8 Preventions of Chickenpox:

Because chickenpox is airborne and very contagious before the rash even appears, it is difficult to avoid. It is possible to catch chickenpox from someone on a different angle in the supermarket, who does not even know they have chickenpox.

A chickenpox vaccine is part of the routine immunization schedule.

1. Children receive two doses of the traditional chickenpox vaccine. The first should be given when the child is 12 – 15 months old. Children should receive the second dose when they are 4-6 year old.

2. People ages 13 and older who have not received the vaccine and have not had chickenpox should get two doses, 4 – 8 weeks apart.

Almost no one will develop moderate or severe chickenpox if they have received the chickenpox vaccine. The small number of children who do develop chickenpox after they have received the vaccine have only a mild case.

The chickenpox vaccine does not require a booster later in life. However, a similar but different vaccine given later in life may reduce the incidence of herpes zoster (shingles).

Talk to your doctor if you think your child might be at high risk for complications and might have been exposed. Immediate preventive measures may be important. Giving the vaccine early after exposure may still reduce the severity of the disease.

CHAPTER NINE

DISEASES ASSOCIATED WITH WATER

Water is a very essential compound to human health, and it's required for every day life. Despite the above merit, water is also a source of infection. It harbour micro-organisms that are capable of transmitting infections to humans and animals.

Waterborne diseases are pathogenic organisms that give rise to unhealthy conditions or adverse effects on human health, such as death, disability, illness or disorders. These diseases can be spread while bathing, washing, drinking water, or by eating food exposed to contaminated water. These organisms are highly important issues considered pressing in primary health care setting among rural areas in developing countries all over the world. The commonest symptoms recognized to be are Diarrhea and vomiting, while other symptoms reportedly include skin, ear, respiratory, or eye problems.

CLASSIFICATION OF DISEASES ASSOCIATED WITH WATER

- i. Water-borne diseases:**
 - Bacterial in origin e.g. cholera, typhoid fever
 - Viral in origin e.g. hepatitis A and E
 - Protozoan in origin e.g. entomoebiasis
- ii. Water-washed diseases:**
 - Infection of the intestinal tract e.g. cholera
 - Infection of the skin e.g. scabies,
 - Infection of the eyes e.g. trachoma

iii. Water-based diseases:

Infection by contact e.g. Schistosomiasis
 Infection by ingestion e.g. Guinea-worm

iv. Water-related insect vector diseases:

E.g. Malaria, African sleeping sickness, Onchocerciasis.

Water-borne diseases (Bacterial in origin)**i) Cholera:**

Cholera is a Greek word which means the gutter of the roof. It is caused by bacteria- *Vibrio cholerae*, which was discovered in 1883 by Robert Koch during a diarrheal outbreak in Egypt. *V. cholerae* have 2 major biotypes: classical biotype and El Tor which was first isolated in Egypt in 1905. Currently, El Tor is the predominant cholera pathogen worldwide.

The organism is a comma-shaped, gram-negative, aerobic bacillus whose size varies from 1-3 µm in length by 0.5-0.8 µm in diameter. Its antigenic structure consists of a flagella H antigen and a somatic O antigen. It is the differentiation of the latter that allows for separation into pathogenic and nonpathogenic strains.

Epidemiology:

Since 1817, there have been 7 cholera pandemics. The first 6 occurred from 1817-1923 and were caused by *V. cholerae*, the classical biotype. The pandemics originated in Asia with subsequent spread to other continents. The seventh pandemic began in Indonesia in 1961 and affected more countries and continents than the previous 6 pandemics. It was caused by *V. cholerae* El Tor. In October 1992, an epidemic of cholera emerged from Madras, India as a result of a new serogroup (O139). Some experts regard this as an eighth pandemic. This Bengal strain has now spread throughout Bangladesh, India, and neighbouring countries in Asia. Crowding and gathering of people during religious rituals (e.g. Muslims pilgrimage to Mecca or Hindu swimming festivals in holy rivers) enhance the spread of infection. Index cases when travelled back to their homes may pass the organism to at risk individuals leading to secondary epidemic or small scale infection.

Transmission: Cholera is transmitted by the fecal-oral route through contaminated water and food. Person to person infection is rare. The infectious dose of bacteria required to cause clinical disease varies with the source. If ingested with water the dose is in the order of 10³-10⁶ organisms. When ingested with food, fewer organisms are required to produce disease.

Clinical presentation:

Symptoms begin with sudden onset of watery diarrhea, which may be followed by vomiting. Fever is typically absent. The diarrhea has fishy odour in the beginning, but became less smelly and more watery over time.

Management:

Surveillance/ Lab investigation

Cholera surveillance should be part of an integrated disease surveillance system that includes feedback at the local level and information-sharing at the global level.

Cholera cases are detected based on clinical suspicion in patients who present with severe acute watery diarrhoea. The suspicion is then confirmed by identifying *V. cholerae* in stool samples from affected patients. Detection can be facilitated using rapid diagnostic tests (RDTs), where one or more positive samples triggers a cholera alert. The samples are sent to a laboratory for confirmation by culture or PCR. Local capacity to detect (diagnose) and monitor (collect, compile, and analyse data).

Organism can be seen in stool by direct microscopy after gram stain and dark field illumination is used to demonstrate motility. Cholera can be cultured on special alkaline media like triple sugar agar or TCBS agar. Serologic tests are available to define strains, but this is needed only during epidemics to trace the source of infection. Dehydration leads to high blood urea and serum creatinine. Hematocrit and WBC will also be high due to hemoconcentration. Dehydration and bicarbonate loss in stool leads to metabolic acidosis with wide-anion gap. Total body potassium is depleted, but serum level may be normal due to effect of acidosis.

PHARMACOTHERAPY

Fluid therapy: The primary goal of therapy is to replenish fluid losses caused by diarrhea and vomiting. Fluid therapy is accomplished in 2 phases: rehydration and maintenance. Rehydration should be completed in 4 hours and maintenance fluids should replace ongoing losses and provide daily requirement.

Cholera is an easily treatable disease. The majority of people can be treated successfully through prompt administration of oral rehydration solution (ORS). The WHO/UNICEF ORS standard sachet is dissolved in 1 litre (L) of clean water. Adult patients may require up to 6 L of ORS to treat moderate dehydration on the first day.

- Severely dehydrated patients are at risk of shock and require the rapid administration of intravenous fluids. These patients are also given appropriate antibiotics to diminish the duration of diarrhoea, reduce the volume of rehydration fluids needed, and shorten the amount and duration of *V. cholerae* excretion in their stool.
- Mass administration of antibiotics is not recommended, as it has no proven effect on the spread of cholera may contribute to antimicrobial resistance.
- Rapid access to treatment is essential during a cholera outbreak. Oral rehydration should be available in communities, in addition to larger treatment centres that can provide intravenous fluids and 24 hour care. With early and proper treatment, the case fatality rate should remain below 1%.

Drug therapy:

The goals of drug therapy are to eradicate infection, reduce morbidity and prevent complications. The drugs used for adults include tetracycline, doxycycline, cotrimoxazole & ciprofloxacin. For children erythromycin, cotrimoxazole and furazolidone are the drugs of choice.

Zinc is an important adjunctive therapy for children under 5, which also reduces the duration of diarrhoea and may prevent future episodes of other causes of acute watery diarrhoea.

Water and sanitation interventions

The long-term solution for cholera control lies in economic development and universal access to safe drinking water and adequate sanitation. Actions targeting environmental conditions include the implementation of adapted long-term sustainable WASH solutions to ensure use of safe water, basic sanitation and good hygiene practices in cholera hotspots. In addition to cholera, such interventions prevent a wide range of other water-borne illnesses, as well as contributing to achieving goals related to poverty, malnutrition, and education. The WASH solutions for cholera are aligned with those of the Sustainable Development Goals.

Community Engagement

Community Engagement means that people and communities are part of the process of developing and implementing programmes. Local culture practices and beliefs are central to promoting actions such as the adoption of protective hygiene measures such as hand washing with soap, safe preparation and storage of food and safe disposal of the faeces of children. Funeral practices for individuals who die from cholera to prevent infection among attendees.

Community engagement continues throughout outbreak response with increased communication regarding potential risks, symptoms of cholera, precautions to take to avoid cholera, when and where to report cases and to seek immediate treatment when symptoms appear. The communities should be part of developing programmes to address needs including where and when to seek treatment.

Oral cholera vaccines

Currently there are three WHO pre-qualified oral cholera vaccines (OCV): Dukoral, Shanchol, and Euvichol-Plus. All three vaccines require two doses for full protection.

Dukoral is administered with a buffer solution that, for adults, requires 150 ml of clean water. Dukoral can be given to all individuals over the

age of 2 years. There must be a minimum of 7 days, and no more than 6 weeks, delay between each dose. Children aged 2 -5 require a third dose. Dukoral® is mainly used for travellers. Two doses of Dukoral provide protection against cholera for 2 years.

Shanchol and Euvichol-Plus have the same vaccine formula, produced by two different manufacturers. They do not require a buffer solution for administration. They are given to all individuals over the age of one year. There must be a minimum of two weeks delay between each dose of these two vaccines. Two doses of Shanchol™ and Euvichol-Plus® provide protection against cholera at least for three years, while one dose provides short term protection.

Shanchol is prequalified to be used in a Controlled Temperature Chain, an innovative approach to vaccine management allowing vaccines to be kept at temperatures outside of the traditional cold chain of +2°C to +8°C for a limited period of time under monitored and controlled conditions.

Complications:

If dehydration is not corrected adequately and promptly it can lead to hypovolemic shock, acute renal failure and death. Electrolyte imbalance is common. Hypoglycemia also occurs in children. Complications of therapy like over hydration and side effects of drug therapy are rare.

Prevention and control:

- i) Education on hygiene practices.
- ii) Provision of safe, uncontaminated, drinking water to the people.
- iii) Antibiotic prophylaxis to house-hold contacts of index cases.
- iv) Vaccination against cholera to travelers to endemic countries & during public gatherings.

9.3 Water-borne diseases (Viral in origin)

i) Hepatitis A

Improvements in hygiene, public health policies, and sanitation had the greatest impact on this disease, while vaccination and passive immunization have successfully led to some reduction in illness in high-risk groups. Reduced encounters with HAV at a young age have

resulted in both a decline in herd immunity and a change to the epidemiology of the illness, with increases in the mean age of occurrence of illness attributed to acute HAV in western societies. Although this may lay a framework for potential epidemics in the future, public health policies and newly implemented immunization practices are likely to reduce this potential.

Epidemiology:

Hepatitis A has a worldwide distribution. The highest seropositivity (antibody to HAV) is observed in adults in urban Africa, Asia, and South America, where evidence of past infection is nearly universal.

Acquisition in early childhood is the norm in these nations and more often than not is asymptomatic. Factors that predispose to early acquisition include overcrowding, poor sanitation, social practices, and lack of a reliable clean water resource. Within the socioeconomic framework (i.e., class structure) of some developing nations, exists differing frequencies of HAV antibody in the older population and, accordingly, sporadic cases may be observed in some individuals.

Until recently, US CDC data supported cycles of disease occurring every 5-10 years. Some of these outbreaks correlated with the wars of the 20th century, where people returned from areas of high endemicity. In recent years, this pattern has disappeared and has been associated with a decline in overall incidence of new infection.

In Shanghai in 1988, a large shellfish-related epidemic occurred. This provided a unique opportunity to study the incubation and natural history of acute HAV in a large population.

Patients may have mild flulike symptoms of anorexia, nausea and vomiting, fatigue, malaise, low-grade fever (usually not higher than 39.5°C), myalgia, and mild headache.

Smokers often lose their taste for tobacco similar to those presenting with appendicitis.

Clinical features:**Icteric phase:**

Dark urine appears first (bilirubinuria). Pale stool soon follows; however, this is not universal. Jaundice occurs in most (70-85%) adults infected with acute HAV. Jaundice is less likely in children and is uncommon in infants. The degree of icterus also increases with age. Abdominal pain occurs in approximately 40% of patients. Itch (pruritus), although less common than jaundice, generally is accompanied by jaundice. Arthralgias and skin rash, although associated, are less frequent than the above symptoms. The rash more often occurs on the lower limbs and may have a vasculitic appearance.

Laboratory investigations:

The diagnosis of acute HAV infection is based on serologic testing for IgM antibody to HAV. Test results for IgM anti-HAV are positive at the time of onset of symptoms and usually accompany the first rise in alanine aminotransferase (ALT). This test is sensitive and specific and remains positive for 3-6 months after the primary infection and for as long as 12 months in 25% of patients (Mandy, 1928). False-positive results are uncommon and should be suspected in the event that anti-HAV IgM persists. IgM persists in those with relapsing hepatitis for the duration of this pattern of disease.

Anti-HAV IgG appears soon after IgM and generally persists for many years. The presence of anti-HAV IgG in the absence of IgM indicates past infection or vaccination rather than acute infection. IgG provides protective immunity.

Rises in ALT and aspartate aminotransferase (AST) assays are sensitive for this disease. Levels may exceed values of 10,000 mIU/mL, with ALT levels generally greater than AST levels. Levels usually return to normal over 5-20 weeks. Rises in alkaline phosphatase accompany the acute disease and may progress during the cholestatic phase of the illness. They follow the rises in transaminases.

Bilirubin rises soon after the onset of bilirubinuria and follows rises in ALT and AST levels. Levels may be impressively high and can remain elevated for several months; persistence beyond 3 months is indicative

of cholestatic HAV infection. Higher levels of bilirubin occur in older individuals. Both direct and indirect fractions increase due to hemolysis often occurring in acute HAV infection. Modest falls in serum albumin may accompany the illness.

Prothrombin time usually remains within or near the reference range. Significant rises should raise concern and support closer monitoring. In the presence of encephalopathy, an elevated prothrombin time has ominous implications (e.g., fulminant hepatic failure).

A mild lymphocytosis is not uncommon. Pure red cell aplasia and pancytopenia rarely may accompany infection. Indices of low-grade haemolysis are not uncommon.

Management:

For acute cases, therapy generally is supportive, with no specific treatment for acute uncomplicated illness. Locating the primary source and preventing further outbreaks are paramount. Initial therapy often consists of bed rest. The patient probably should not work during the acute phase of the illness.

Nausea and vomiting are treated with antiemetic

Dehydration may require hospital admission and intravenous fluids.

In most instances, hospitalization is not necessary. Most children have minimal symptoms; adults are more likely to require more intensive care, including hospitalization.

Between 3-8% of cases of fulminant hepatic failure are caused by HAV; however, only 1-2% of HAV infections in adults lead to fulminant hepatic failure.

Tylenol may be administered but with caution and with strict limitations applied to maximum dose (3-4 g/d).

Other treatments are directed by the specific complications.

Complications:

Generally, there are no lasting sequelae of HAV infection. Fatalities are rare; occurring in less than 0.2% of cases. Fatality is more frequent in elderly patients and in those with underlying liver disease. In children, liver transplantation has been performed for fulminant hepatic failure.

In France, 10% of cases of fulminant failure in children are caused by HAV. The outcomes from transplantation are the same as for others with fulminant disease. Recurrent disease does not occur following transplantation despite immunosuppression. Prolonged cholestasis may follow the acute infection. The frequency at which this occurs increases with age. Prolonged cholestasis is characterized by a protracted period of jaundice (longer than 3 months) and resolves without intervention. Corticosteroids and ursodeoxycholic acid may shorten the period of cholestasis. The usual features of cholestatic viral hepatitis A are pruritus, fever, diarrhea, and weight loss, with serum bilirubin levels greater than 10 mg/dL. Some believe that the use of corticosteroids may predispose patients to developing relapsing hepatitis A. Good data to support this are lacking.

Acute renal failure, interstitial nephritis, pancreatitis, red cell aplasia, agranulocytosis, bone marrow aplasia, transient heart block, Guillain-Barré syndrome, acute arthritis, Still disease, lupus-like syndrome, and Sjögren syndrome have been reported in association with HAV. These complications are all rare.

An entity that has received important discussion in the literature is autoimmune hepatitis following HAV infection. A postulated mechanism involves molecular mimicry and genetic susceptibility, much in the same way as that proposed in type 1 diabetes. Steroid therapy for this condition was associated good clinical response and improvement in biochemical and clinical parameters in a similar way to traditional autoimmune hepatitis. This is, however, confined to isolated case reports and the results of larger clinical trials are not available.

Relapsing hepatitis A occurs in 3-20% of patients with acute hepatitis A and uncommonly takes the form of multiple relapses. Following a typical acute course of HAV infection, a remission phase occurs, with partial or complete resolution of clinical and biochemical manifestations. The initial flare usually lasts 3-6 weeks and relapse occurs after a short period (usually <3 wks.) and mimics the initially presentation, although it usually is clinically milder. There is a tendency to greater cholestasis in these patients. Vasculitic skin rashes and

nephritis may be additional clinical clues to this syndrome. During relapses, shedding of virus can be detected. IgM antibody is positive. The clinical course is toward resolution with the period between flares increasing. The total duration is between 3-9 months.

Liver transplantation has been performed in patients with this condition where signs of significant decompensation have occurred. Corticosteroid treatment has been shown to benefit the clinical course, although, without treatment, the course, in itself, is generally benign.

Prognosis:

Prognosis is excellent. Long-term immunity accompanies infection. Recurrence and chronic hepatitis does not occur.

ii. Hepatitis E:

Hepatitis E virus (HEV) is an enterically transmitted self-limited infection. It is spread by faecally contaminated water within endemic areas. Outbreaks can be epidemic and individual. It has many similarities with hepatitis A. The incubation period ranges from 15 days to 60 days, and the course of infection has 2 phases termed prodromal and icteric.

Prodromal-phase symptoms include the following:

Myalgia

Arthralgia

Fever with mild temperature elevations (25-97%)

Anorexia (66-100%)

Nausea/vomiting (30-100%)

Weight loss (typically 2-4 kg)

Dehydration

Right upper quadrant pain that increases with physical activity

Icteric-phase symptoms include the following:

Jaundice - May be difficult to see with some patients' natural skin color; serum bilirubin level is greater than 3 mg/dL; scleral icterus is present

Dark urine

Light-colored stools (20-40%)

Pruritus (50%)

Other features include the following:

Urticarial rash

Diarrhea

Rapidly increasing serum amino transferase (alanine aminotransferase (ALT), aspartate aminotransferase (AST) levels that peak within 4-6 weeks of onset and gradually decrease to normal within 1-2 months

Viral excretion in stool persisting 14 days from onset

Clinical features of HEV are similar to other hepatitises and include the following:

Abdominal pain (35-80% of patients)

Jaundice

Anorexia

Hepatomegaly (10-85%)

Malaise (95-100%)

Vomiting

When or how long the patient is infectious cannot be determined, but infectivity may relate to the presence of the virus in the stool.

Lab investigation:

Diagnostic tests for HEV are not available commercially.

Management:

Therapy should be predominantly preventive, relying on clean drinking water, good sanitation, and proper personal hygiene. Travelers to endemic areas should avoid drinking water or other beverages that may be contaminated and should avoid eating uncooked shellfish. Care should be taken while preparing uncooked fruits or vegetables. Boiling water may prevent infection, but the effectiveness of chlorination is unknown.

No immunoprophylaxis is available. Immunoglobulin from infected patients is not effective in preventing outbreaks or sporadic cases.

Prototype vaccines are being developed using animal models. To date, this is hindered by an inability to maintain the virus in cell cultures. Once infection occurs, therapy is limited to support. Provide patients with adequate hydration and electrolyte repletion. Hospitalization is indicated only for patients unable to maintain oral intake.

Prevention and control:

Control at the source, with treatment of contacts to prevent, where possible, further cases of disease is the primary goal. Long-term secondary goals include immunization, which increases herd immunity and reduces the likelihood of further outbreaks in high-risk communities. Education about transmission and prevention of transmission (e.g., hand washing, safe food sources) also is important.

9.4 Water-Borne Diseases (Protozoan Origin)

i. Amoebiasis

Amoebiasis is an infection caused by the protozoal organism *Entamoeba histolytica* and includes amebic colitis and liver abscess. In developed countries, infection occurs primarily among travellers to endemic regions, recent immigrants from endemic regions, homosexual males, immunosuppressed persons, and institutionalized individuals. Transmission usually occurs by food-borne exposure, particularly when food handlers are shedding cysts or food is cultivated in feces-contaminated soil, fertilizer, or water. Less common means of transmission include contaminated water, oral and anal sexual practices, and direct rectal inoculation through colonic irrigation devices.

Epidemiology:

Entamoeba species infect approximately 10% of the world's population. The prevalence of infection is as high as 50% in areas of Central and South America, Africa, and Asia. Amebic liver abscess is 7-12 times more common in men than in women, although the sex distribution is equal in children. Amebic colitis affects both sexes equally. Young children appear to be at higher risk for fulminant invasive disease, resulting in a higher mortality rate.

Clinical presentation:

Patients with amebic colitis typically present with a history of several weeks of abdominal pain, diarrhea, and bloody stools. Fever is uncommon and occurs in approximately 10-30% of patients. Because of the gradual onset of disease, weight loss is a common complaint and may be accompanied by symptoms of volume depletion (e.g., orthostasis). Uncommon manifestations include amoeba, fulminant colitis, and rectovaginal fistulas.

Fulminant or necrotizing colitis is the most serious manifestation, which occurs in approximately 0.5% of patients and has a mortality rate of greater than 40%. Predisposing factors for fulminant colitis include poor nutrition, pregnancy, corticosteroid use, and very young age.

Patients with amebic liver abscess typically present with a 1- to 2-week history of fever and right upper quadrant abdominal pain. Unlike amebic colitis, amebic liver abscess is associated with fever in 85-90% of patients. Patients with a single abscess may be more likely to present subacutely, with prominent weight loss, and fewer than half the patients have fever and abdominal pain. Most patients with liver abscess (i.e., 60-70%) do not have concomitant colitis, although a history of dysentery within the previous year may be obtained. History of alcohol abuse is common, but how this condition may contribute to the development of a liver abscess still is unclear.

Pleuropulmonary amoebiasis is a rare but serious complication of amebic liver abscess, usually caused by rupture of a superior right upper lobe abscess with erosion through the diaphragm. Patients with this complication present with cough, pleuritic chest pain, dyspnea, and, occasionally, necrotic sputum. Intraperitoneal rupture of amebic liver abscess occurs in 2-7% of patients. These patients can present with a rigid abdomen that may be diagnosed erroneously as a perforated viscus.

Cerebral amoebiasis is a rare cause of brain abscess and is characterized by an abrupt onset of mental status change and/or focal neurologic deficits. Progression to death occurs over 12-72 hours without adequate therapy.

Lab investigation:**Microscopy:**

Microscopic examination of a single stool specimen from a patient with amebic colitis is only 33-50% sensitive. Results on a repeated stool examination in patients with proven amebic liver abscess are positive in 8-40% of cases.

Identification of the parasite in a liver abscess aspirate is only 20% sensitive.

The World Health Organization (WHO) recommends that intestinal infection be diagnosed with an *E histolytica*-specific test, thus rendering the classic stool ova and parasite examination obsolete in this setting; however, finding quadrinucleated cysts or trophozoites containing ingested erythrocytes in stool is considered by many to be diagnostic for amebic colitis.

Antigen detection:

A stool antigen test specific for *E histolytica* is available from Laboratories (Blacksburg, Va). This test uses monoclonal antibodies specific for the galactose (Gal)/N-acetyl-D-galactosamine (GalNAc) lectin of *E histolytica* and has a sensitivity of 87% and specificity of greater than 90% compared with culture. Early data on antigen detection tests for serum and liver abscess aspirates are promising.

Serology:

Serum antiamebic antibody tests are an important adjunct to antigen detection, particularly in cases of amebic liver abscess, in which the parasite is found less commonly in the stool. Tests for antibodies to amoeba are 90% sensitive for amoebic liver abscess and 70% sensitive for amebic colitis. A problem with serological tests is that the antibody persists for years after the initial infection; therefore, differentiating

between current and previous infection may be difficult, especially in individuals from endemic areas.

Polymerase chain reaction:

Preliminary results of polymerase chain reaction (PCR) and DNA probes for strain-specific detection of *E histolytica* in stool and liver abscess aspirates appear encouraging. Sensitivity when performed on stool samples is estimated at 87%.

Management:

Most individuals with *E histolytica* infection may be treated on an outpatient basis. Exceptions include the following:

1. Patients with severe colitis requiring intravenous volume replacement
2. Patients with fulminant colitis that may require surgical intervention
3. Patients with liver abscess of uncertain etiology or not responding to therapy
4. Patients with suspected liver abscess rupture

Metronidazole (Flagyl):

Active against various anaerobic bacteria and protozoa, appears to be absorbed into cells. Intermediate metabolized compounds are formed and bind DNA and inhibit protein synthesis, causing cell death. Antimicrobial effect may be due to production of free radicals. It is indicated for invasive *E histolytic* infections.

Prevention and control:

Prevention of amoebiasis is accomplished by eradicating fecal contamination of food and water through improved sanitation, hygiene, and water treatment. Amebic cysts are not killed by soap or low concentrations of chlorine or iodine; therefore, when in an area of endemicity, water should be boiled and vegetables should be washed with a detergent soap and soaked in acetic acid or vinegar for 10-15 minutes before consumption.

Avoiding sexual practices that allow fecal-oral contact may reduce the risk of sexual transmission of infectious cysts.

Screen family members or close contacts of an index case.

Efforts to develop a vaccine are underway but have been complicated by an incomplete understanding of the mechanisms of immunity in humans and the lack of well-studied intestinal models of infection.

Complications:

Amoebic colitis

Fulminant or necrotizing colitis

Toxic megacolon

Amoeboma

Rectovaginal fistulas

Amebic liver abscess

Intrathoracic or intraperitoneal rupture with or without secondary bacterial infection

Direct extension to pleura or pericardium

Brain abscess

Prognosis:

The prognosis of cure following treatment for invasive amoebiasis is good; however, prior infection and treatment do not protect against future colonization or recurrent invasive disease.

9.5 Water-Based Diseases (Infection By Contact):

i) Schistosomiasis

After malaria, Schistosomiasis is the second most prevalent tropical disease in the world. In honor of Theodore Bilharz, in some parts of the world it is also known as bilharzia. He first identified the etiological agent for *Schistosoma hematobium* in Egypt in 1851.

At-risk persons include those who live or travel in areas where Schistosomiasis occurs and who come into contact with fresh water where the appropriate type of snail intermediate host is present. The main forms of human Schistosomiasis are caused by 5 species of flatworm in the genus *Schistosoma*, within the class trematode. The 5

species are as follows: *S. hematobium*, *S. mansoni*, *S. japonicum*, *S. intercalatum*, and *S. mekongi*. The worms are also called blood flukes because they live in the vascular system of humans and other vertebrates.

The life cycle of the flatworms that cause human Schistosomiasis involves a sexual stage in the human and an asexual stage in the fresh water snail host. The adult worms are small, 12-26 mm long and 0.3-0.6 mm wide, and vary with the different species. *S. hematobium* lives in the venous plexus near the urinary bladder and ureters, *S. mansoni* lives in the inferior mesenteric vein, and *S. japonicum* lives in the superior mesenteric vein of both the large and small intestines.

Adult worms mate and lay eggs. The eggs are non-operculate, possess a spine, and contain a miracidium. The microscopic appearance of the egg allows diagnostic differentiation of the 5 species. An adult *S. hematobium* produces 20-200 round, terminally spined eggs per day, *S. mansoni* produces 100-300 ovoid, laterally spined eggs per day, and *S. japonicum* produces 500-3500 round, small, laterally spined eggs per day. The eggs of *S. intercalatum* have prominent, terminal spines and *S. mekongi* have small, lateral spines.

When the ova reach the fresh water, the miracidia are released and they penetrate the snail. Within 3-5 weeks, they asexually multiply into hundreds of fork-tailed cercariae. The cercariae leave the snail and swim to a human or non-human animal where they penetrate the skin. Once inside, cercaria travel to the heart, the lungs, and through the systemic circulation to reach the portal veins where they develop into an adult worm. The time between cercariae penetration and the first ova production is 4-6 weeks.

Clinical features:

Symptoms depend on the species of schistosome infection, the duration and severity of infestation, and the immune response to the eggs. Typically, onset is insidious. *S. mansoni*, *S. mekongi*, *S. intercalatum*, and *S. japonicum* cause intestinal tract and liver disease. *S. hematobium*

only rarely causes intestinal or liver disease, but characteristically causes urinary tract disease.

Hepatic Schistosomiasis –

In the early stage there is dyspepsia, flatulence, and pain in the left hypochondrium due to the enlargement of the spleen. Anemia or cor pulmonale may cause generalized pain, weakness, and shortness of breath. In the later stages, abdominal distention, lower limb edema, hematemesis, and melena can occur. Symptoms of liver failure are rare unless other infectious, toxic, or malignant causes of hepatitis are present.

Intestinal Schistosomiasis:

Fatigue, abdominal pain, diarrhea, and dysentery

Urinary Schistosomiasis:

Dysuria, urinary frequency, and terminal hematuria

Cardiopulmonary Schistosomiasis:

May cause larval pneumonitis with a cough, mild wheezing, and a low-grade fever.

Schistosomal cor pulmonale:

Easy fatigability, palpitations, dyspnea on exertion, and hemoptysis

Central nervous system Schistosomiasis:

Focal and generalized seizures; headache; and myeloradiculopathy with lower limb and back pain, bladder dysfunction, paresthesia, and lower limb weakness

Stool or urine analysis:

Aimed at identify and specifying the eggs in the stool or urine. Direct stool examination is not a sensitive test and urinary excretion of eggs is not uniform. The urine is most likely to be positive for *S. hematobium* between 10AM-2PM. Quantification of the egg excretion is calculated by collecting 24-hour urine or stool, homogenizing the sample, and

counting the eggs in a measured sample. Urine or stool egg count in a 24-hour collection will quantitate the severity of the infection. Less than 100 eggs per gram is a light infection, 100-400 eggs per gram are moderate and greater than 400 eggs per gram is heavy.

Egg viability test:

Important for assessing the effectiveness of treatment. It requires mixing the stools or urine with room temperature distilled water and observing for hatching miracidia. An active infection will give viable eggs, while treated or past infection will result in non-viable eggs and an absence of miracidia.

Acute illness is often associated with eosinophilia in the blood and tissues. With chronic illness, peripheral eosinophilia may be minimal or absent while tissue eosinophilia persists.

Urinary Schistosomiasis (occurs with chronic disease).

Urine syringe filtration techniques will give a quantitative estimate of eggs in the urine. Urine analysis and culture for hematuria, proteinuria, leukocyturia, and associated urinary infections

A Salmonella urinary tract infection should always make the clinician suspect Schistosomiasis.

Blood chemistries, including renal function tests:

Liver function tests will usually be normal until the end stage of disease. An exception may include a mild elevation of alkaline phosphatase. If liver function tests are abnormal, look for other co-infections or diseases.

Management:

The aim of chemotherapy is two-fold. The first is to cure the disease or at least minimize morbidity. The second is to control transmission of the parasite in the endemic areas. Praziquantel and oxamniquine (no longer available in the United States) are commonly used, but Praziquantel is the treatment of choice for all species of Schistosomiasis.

Prevention and control:

No vaccine or prophylactic chemotherapy is available. Travelers to endemic areas should avoid contact with fresh water. Suspect acute Schistosomiasis in a setting of recent fresh water contact, and treat early if diagnostic tests are positive or clinical suspicion is high.

Controlling Schistosomiasis in an endemic area should include the following:

Population-based chemotherapy

Providing a safe water supply

Health education includes improving water sanitation and avoiding schisto-contaminated urine or stool.

Snail control

Complications:

GI bleeding

GI obstruction

Malnutrition

Schistosomal nephropathy

Renal failure

Pyelonephritis

Bladder cancer

Sepsis (*Salmonella*)

Pulmonary hypertension

Cor pulmonale

Neuroschistosomiasis

Prognosis:

Early disease usually improves with treatment, so also is hepatic, renal, and intestinal pathology. Hepatosplenic Schistosomiasis carries a relatively good prognosis because hepatic function is preserved until the end of the disease (unless variceal bleeding occurs). Cor pulmonale usually does not significantly improve with treatment. Depending on location and size, brain lesions usually improve with treatment but

spinal cord Schistosomiasis carries a guarded prognosis. Praziquantel should be given as soon as possible.

9.6 Water-related insect vector diseases:

1. Malaria

Predominantly observed in the tropics, malaria is a potentially life-threatening disease that may present with fever and a wide range of symptoms. Humans are infected when bitten by the female *Anopheles* mosquito vector, resulting in the transmission of this protozoan organism.

Four species can cause disease, specifically, *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale*, and *Plasmodium malariae*. Timely diagnosis of the correct species is required especially to identify *P falciparum*, which can be fatal and often is resistant to standard chloroquine treatment. Occasionally, patients may be infected with more than one species. *P falciparum* and *P vivax* are responsible for the majority of new infections. Each species has a defined area of endemicity, though geographical overlap is common. Species can usually be distinguished by morphology on a blood smear.

Malaria in travelers typically presents weeks after patients leave the endemic area. In some patients, the disease presents months to years later; therefore, history of even a remote exposure to an endemic area should be elicited. The symptoms of malaria are nonspecific, and since timely diagnosis and treatment are required, malaria should be considered in all patients from the tropics who present with fever.

Patients typically acquire malaria in an endemic area after a mosquito bite. Cases secondary to airport malaria and infection secondary to transfusion of infected blood are extremely rare. The risk of acquiring infection depends on the intensity of malaria transmission and use of precautions (e.g., bed nets, DEET [diethyltoluamide], malaria prophylaxis). After a mosquito takes a blood meal, the malarial sporozoites enter hepatocytes rapidly, i.e., within minutes, and emerge into the bloodstream after a few weeks. These merozoites rapidly enter erythrocytes and develop through the erythrocytic life cycle, from

trophozoites through schizonts. Rupture of the infected erythrocytes results in fever and the release of merozoites.

These continue the cycle logarithmically increasing the parasite burden. A small percentage of the parasites become gametocytes, which undergo sexual reproduction when taken up by the mosquito, develop into infective sporozoites, and continue the transmission cycle.

Each species has a specific incubation period. *P vivax* and *P ovale* have a hypnozoite form, for which the parasite can linger in the liver for months before emerging and recur after an initial infection. Treating the hypnozoite form with a second agent in addition to the blood stage form in two species is critical because these patients may experience relapses. Omitting the treatment for the hypnozoite form for these species is a common error in treatment of malaria.

Severe disease typically is secondary to *P falciparum*. This species is more virulent due to the high parasitemias that can occur and the property of sequestration, which may contribute to end organ damage. Sequestration is a specific property of this species, which may provide a clue to its identification. As it develops through the 48-hour life cycle it demonstrates adherence properties, which result in the sequestration of the parasite into small postcapillary vessels. For this reason, only early forms are seen in the peripheral blood, before the sequestration property develops and this is an important diagnostic clue to this species.

These sequestration phenomena may contribute to mental status changes and coma, seen exclusively in *P falciparum*. In addition, cytokines and a high burden of parasites contribute to the end organ disease. End organ disease may rapidly develop with *P falciparum* and specifically involves the CNS, lungs, and kidneys. These severe manifestations may occur in the non-immune traveler or young children who live in the endemic area.

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Epidemiology:

Forty percent of the world's population live in endemic areas and are at risk for acquiring this infection. It is estimated that 300-500 million infections develop each year.

Clinical features:

Patients typically become symptomatic a few weeks after infection, though the host's previous exposure or immunity to malaria affects symptomatology and incubation periods. In addition, each species of malaria has a typical incubation period.

P. malariae does not have a hypnozoite stage, but patients may have a prolonged, asymptomatic, erythrocytic infection, which becomes symptomatic years after leaving the endemic area. Severity of illness is affected by previous exposure to malaria and patient age. In addition, various genetic factors may enhance or limit disease, such as protective factors of sickle cell disease, hemoglobinopathies, and polymorphisms in the host's TNF gene.

The periodicity of fevers associated with each species (i.e., 48 hours *P. falciparum*, *P. vivax*, *P. ovale*; 72 hours *P. malariae*) is not apparent during initial infection because of multiple broods emerging in the blood stream. Long-standing synchronous infections are more likely to present with the classical fever patterns. In general, the periodicity of fevers cannot be relied on when considering this diagnosis.

The symptoms of malaria infection are nonspecific and may manifest as a flulike illness with fever, headache, malaise, fatigue, and muscle aches. Some patients may present with diarrhea and other GI symptoms. Immune individuals may be completely asymptomatic or present with mild anemia. Non-immune patients may quickly become very ill. Severe malaria is primarily secondary to *P. falciparum*, though death from splenic rupture has been reported in non-*P. falciparum* malaria.

Severe malaria:**Cerebral malaria:**

Coma may occur. Usually, the coma can be distinguished from a postictal state secondary to generalized seizure. In evaluating patients

with coma complicating malaria, hypoglycemia and other CNS infections should be excluded.

Severe anemia:

The anemia is multifactorial and usually is associated with *P falciparum* infection. In non-immune patients, the anemia may be secondary to erythrocyte infection and loss of infected RBCs. Also, uninfected RBCs are inappropriately cleared, and bone marrow suppression may be involved.

Renal failure:

This is a rare complication of malarial infection. Infected erythrocytes adhere to the microvasculature in the renal cortex, often resulting in oliguric renal failure. Typically, renal failure is reversible, and dialysis often is needed to support patients until kidney function recovers. Chronic infection with *P malariae* may rarely result in nephrotic syndrome.

Pulmonary edema:

Pulmonary vasculature pressures are normal, which suggests the pulmonary edema is secondary to capillary leak syndrome. This condition typically occurs in patients with severe malaria.

Lab investigations:

The diagnosis of malaria should be supported by the identification of the parasites on a thin or thick smear of blood. The only rare exception is *P falciparum*, in which it is possible for all the parasites during the life cycle to be sequestered out of the peripheral blood in late stage forms. If no alternative diagnosis is found in an at-risk patient with possible cerebral malaria (i.e., the lumbar puncture is unrevealing) initiating therapy for presumptive malaria, while obtaining further blood smears to make the diagnosis, is reasonable.

Three thick and thin smears 12-24 hours apart should be obtained for the diagnosis of malaria. The highest yield of peripheral parasites is at,

or soon after, a fever spike, but smears should not be delayed awaiting fever spikes.

Thick smears are 20 times more sensitive than thin smears, but speciation may be more difficult. Take finger prick blood and place a drop on a glass slide with the end of a second slide mixing the blood until it clots. Dry thoroughly, and then stain in Giemsa stain buffered to 7.2. Leave on the slide 30 minutes. The World Health Organization recommends that at least 100 fields, each containing approximately 20 WBCs, be screened before calling a thick smear negative. In non-immune patients, there may be a very low parasitemia and a longer evaluation of the slide should be considered.

Thin smears are less sensitive but may allow easier speciation. Smears are prepared by taking a small drip of finger prick blood to one end of a slide and taking a second slide at 45 degree angle and steadily drawing the blood across the slide. The sample should be fixed in methanol and stained with Giemsa stain.

Parasitemia can be determined by counting the number of parasites per 1000 RBC or, if low parasitemia, the number of parasites per 500 WBC, corrected by the total RBC, and WBC counts to give the number of parasites per mL of blood volume.

Alternative diagnostic methods

Alternative diagnostic methods are used particularly if there is not sufficient expertise in reading smears. Alternatively, particularly if there is no expertise available to detect parasites, methods that stain the parasite using acridine orange have been developed. These include the Kawamoto technique, in which blood smears on a slide are stained with acridine orange and examined with either a fluorescence microscope or a light microscope adapted with an interference filter system. This results in a differential staining of nuclear DNA in green and of cytoplasmic RNA in red, which allows recognition of the parasites. A similar technique, the quantitative buffy coat (QBC), is as sensitive as thick smears but cannot speciate. A thin smear needs to be examined.

Monoclonal antibody (AB) to histidine-rich protein-2 appears very sensitive and specific.

In general, blood cultures should be drawn in a febrile patient and often typhoid is in the differential in a patient returning from the tropics. Hypoglycemia may occur with malarial infection and should be ruled out in a patient with mental status changes. Patients should have assessment of hemoglobin (decreased in 25%, often profoundly in young children) and platelet count (thrombocytopenia in 50-68%, and abnormal LFTS in 50%). Importantly, typically fewer than 5% of patients have an elevated WBC count, and if leucocytosis is present, the examiner should entertain a broader differential diagnosis. If the patients are to be treated with primaquine, G-6-PD level should be tested. If the patient has cerebral malaria, blood glucose should be measured to rule out hypoglycemia as a cause of mental status changes.

Management:

Prevention and control:

Avoiding mosquitoes through limiting exposure during typical blood meals (i.e., dawn, dusk) wearing long-sleeved clothing, and use of insect repellants may prevent infection. Adult dose 95% DEET lasts as long as 10-12 hours; 35% DEET lasts 4-6 hours. For children, use concentration of less than 35% DEET. Use sparingly and only on exposed skin. Remove DEET when no longer exposed. Consider using bed netting that is impregnated with permethrin and chemoprophylaxis with antimalarials.

Artemisinin based combination therapy:

Artemisinin is a Chinese product derived from the wormwood plant *Artemisia annua*. Semi-synthetic derivatives including artemether and artesunate are now widely available

1. Artemisinin (500mg tablets) give 10-20 mg/kg on day 1 (500-1,000 mg) orally then 500mg for 4 days. Then give mefloquine 15mg base/kg or split dose 25mg base/kg.
2. Artesunate (50 & 60 mg vials for intravenous use): for severe malaria 120mg I.V. stat. 60 mg at 4, 24 and 48 hours, 50-60 mg on days 3-5. Give mefloquine as above.

3. Dihydroartemisinin (20 mg tablets): First dose 120mg then 60mg daily for 4-6 days then give mefloquine as above.
4. Artemether (vials for intramuscular use): For severe malaria 3.2 mg/kg intramuscularly stat then 1.6mg twice daily for 3-7 days, give mefloquine as above.
5. Artemether plus lumefantrine (Co-artemTM or RiametTM)

Advantages of ACT:

1. High efficacy and rapid clearance of parasites
2. Experience in SE Asia shown to slow down the development of resistance
3. Artemisinin reduces gametocyte carriage thus reduces malaria transmission

CHAPTER TEN

SEXUALLY ASSOCIATED DISEASES 1: BACTERIAL INFECTIONS

Admittedly, sexually transmitted Infections are caused by Bacteria, viruses and Parasites. But in this chapter, I will discuss bacterial infections.

Generally, sexually transmitted diseases (STDs) are contracted or acquired by sexually oriented behavior or contact. These pathogenic microorganisms (The bacteria) may pass from person to person in blood, semen, or vaginal and other bodily fluids.

Sexually transmitted diseaess caused by Bacteria include: Gonorrhea, syphilis and chlamydia

1. **Gonorrhea:**

Aetiology: How is Gonorrhea contracted or causes? Gonorrhea can affect humans, ranging from infants to adult. It's transmitted by direct inoculation of secretions from one mucous membrane to another, most commonly through vaginal, anal or oral sexual intercourse. Infection of the eye most commonly results from autoinoculation by an infected individual. Gonococcal infection among infants usually results from exposure to infected cervical exudates at birth, making screening during pregnancy important. The creen is important because Gonorrhea can also be transmitted from birthing parent to baby during delivery.

Pathophysiology of Gonorrhea: *N. gonorrhoeae* is the responsible bacteria for Gonorrhea. it's known as a widespread sexually transmitted disease caused when *Neisseria gonorrhoeae* bacteria infect the normally protective inner lining of human genital tissues.

This bacteria enters through sexual contact and attach to mucosa and epithelial cells. They invade the cells and damage the mucosa. The body will usually cause an inflammatory response with exudate at the site of infection.

Clinical manifestations of Gonorrhoea: Most times the symptoms of Gonorrhoea are not noticeable. But even someone who is asymptomatic carrier can still transmit gonorrhoea.

The signs and symptoms of the diseases are manifested based on the gender. **For human male**, within 2 to 30 days after exposure, the individual will develop signs and symptoms of Gonorrhoea.

The following are possible evidences of Gonorrhoea:

i. Itching and soreness in the anus ii. Burning or pain during urination may be the first symptom to notice. iii. Urgency of urination iv. Discoloration and swelling at the penis opening v. Testicular swelling or pain vi. Pain when having bowel movements vii. Rectal bleeding or discharge

viii. A pus-like discharge or drip from the penis. This discharge could be yellow, white, beige, or greenish.

For human female:

Many females don't develop any symptoms of gonorrhoea. But those who do experience can appear from a day to several weeks after exposure.

These symptoms might be very similar to that of vaginal yeast or other bacterial infections, which can make them even more difficult to recognize.

Possible symptoms include: a. watery, creamy, or greenish vaginal discharge

b. Pain or burning while urinating. c. an urge to urinate more frequently. d. Heavier periods or spotting between periods. e. Pain during penetrative vaginal sex. f. Sharp pain in the lower abdomen. Itching and soreness in the anus. g. Rectal bleeding or discharge. h. painful bowel movements

Diagnostics findings: There are many strategies employed by health workers to diagnose this condition, but not limited to these:

- i. Urine examination. ii. Fluid emanation: This method involves penis, vagina, throat, or rectum swab to get a sample of fluid for culture.
iii. Blood test: it's important, but rarely used to detect gonorrhoea. It's not usually conclusive.

Therapeutic Management:

Education:

- a. No sex for 7 days. b. Safe sex. c. Condom use provides partial protection. d. Notify sexual partners for treatment. E. Early treatment. f. Allow patient teach-back on symptoms
g. Explain fertility and morbidity risks. h. Educate parents on importance of Erythromycin post-birth

Nursing Intervention

1. Use standard precautions when obtaining specimens for laboratory examination and when caring for the patient.
2. Isolate the patient with an eye infection.
3. If the patient has gonococcal arthritis, apply moist heat to ease the pain in the affected joints.
4. Before treatment, determine if the patient has any drug sensitivities.
5. Monitor the patient for complications.
6. Tell the patient that until cultures prove negative, he's still infectious and should avoid unprotected sexual contact.
7. Urge the patient to inform his sexual partners of his infection so that they can seek treatment.
8. Advise the partner of an infected person to receive treatment even if she doesn't have positive cultures.
9. Counsel the patient and his sexual partners to be tested for human immunodeficiency virus and hepatitis B infection.
10. Instruct the patient to be careful when coming in contact with his bodily discharges so that he doesn't contaminate his eyes.
11. Tell the patient to take anti-infective drugs for the length of time prescribed.
12. To prevent reinfection, tell the patient to avoid sexual contact with anyone suspected of being infected, to use condoms during

intercourse, to wash genitalia with soap and water before and after intercourse.

Medical Management

According to GUM Clinic, National guidelines recommend first line treatment with Ceftriaxone 500 mg by intramuscular (IM) injection as a single dose, plus azithromycin 1 g orally as a single dose.

If an IM injection is contraindicated or refused, oral cefixime 400 mg orally as a single dose, plus azithromycin 1 g orally as a single dose can be offered.

If cephalosporins are contraindicated (for example the person has a true allergy to penicillin-type antibiotics), consider a fluoroquinolone (ciprofloxacin 500 mg, single oral dose or ofloxacin 400 mg, single oral dose) plus azithromycin 1 g, single oral dose.

Treatment with ciprofloxacin or ofloxacin is only recommended if *N. gonorrhoeae* is proven to be sensitive (i.e. culture and sensitivity results are available for the person or recent sexual partners), as there is a high prevalence of quinolone resistance worldwide.

2. SYPHILIS

Aetiology: Syphilis is caused by the bacteria known as *Treponema pallidum*. It can be gotten through direct contact with a syphilis sore on or in another person's body (mouth, penis, vagina, anus)

. This usually happens during sexually oriented behavior or activity, but the bacteria can also get into the body through cuts on the skin or through mucous membranes.

In 1905, German scientists that the bacterium *Treponema pallidum* is responsible for the infection

Admittedly, Syphilis is primarily transmitted sexually (through oral, anal, or vaginal sex, or direct genital-to-genital contact), but Babies can also acquire syphilis if their mother has an untreated infection (congenital syphilis).

The fact that the bacteria that cause syphilis can't live for a long period outside the human body make it possible for Syphilis not to be contracted by:

Using eating utensils, sharing a toilet, and wearing another person's clothing.

In 2020, 133,945 new cases of syphilis (all stages) were reported in the United States, according to the Centers for Disease Control and Prevention (CDC). Syphilis in people with vaginas is rising slightly more than people with penises, though both groups are seeing an uptick in cases overall.

Syphilis has different stages of Infection which include: primary, secondary, latent, and tertiary

Syphilis is most infectious during the first two stages.

When syphilis is in the hidden, or latent, stage, the disease remains active but often doesn't cause symptoms. Tertiary syphilis is the most destructive to health.

Pathophysiology: *T. Pallidum* is the cause of syphilis. The bacteria enter through a small abrasion during intercourse. In stage I *T. Pallidum* multiplies in the epithelial tissue and small chancre forms. The immune system responds. Stage II the bacteria spread to major organ systems. Stage III is the latent phase where it is a silent infection. Transmission is possible even though there are no signs of infection. Stage IV there is destruction to the skin, bone, cardiovascular complications such as heart failure and aneurysms occur. There is a high chance of morbidity in stage IV as the bacteria overwhelms the body.

Clinical manifestations: Syphilis can be challenging to diagnose. Someone can have it without showing any symptoms for years. However, the earlier syphilis is discovered, the better. Syphilis that remains untreated for a long time can cause major damage to important organs, such as the heart and the brain.

At first, the bacterial infection has minimal to no symptoms. As time goes on, the infection progresses to affect multiple systems in your body, which can then have severe effects.

The clinical manifestations of Syphilis depends on the stage the patient is. There are four stages, which are:

1. **Primary syphilis:** This stage occurs about 3 to 4 weeks after an individual has exposed to the bacteria. It begins with a small, round sore called a chancre. A chancre is painless, but it's highly infectious. People may not even notice when they have one. This sore may appear wherever the bacteria entered the body, such as on or inside the mouth, genitals, or rectum. On average, the sore shows up around 3 weeks after infection, but it can take between 10 to 90 days to appear. The sore remains for 2 to 6 weeks. Sometimes the only symptom will be swollen lymph nodes.
2. **Secondary syphilis:** Skin rashes and a sore throat may develop during the second stage of syphilis. The rash won't itch and is usually found on the palms and soles, but it may occur anywhere on the body. Some people don't notice the rash before it goes away. During this stage Other symptoms may include: Weight loss, headaches, hair loss, fatigue, swollen lymph nodes, aching joints, fever.
3. **Latent syphilis:** The third stage of syphilis is the latent, or hidden, stage. The primary and secondary symptoms disappear, and there won't be any noticeable symptoms at this stage. However, the bacteria remain in the body. This stage could last for years before progressing to tertiary syphilis.
4. **Tertiary syphilis:** The last stage of infection is tertiary syphilis. About 14 to 40 percent. Trusted Source of people with syphilis enter this stage. Tertiary syphilis can occur years or decades after the initial infection. Tertiary syphilis can be life-threatening. Some other potential outcomes of tertiary syphilis include: blindness, loss of hearing, mental health conditions, memory loss, destruction of soft tissue and bone, neurological disorders, such as stroke or meningitis, heart disease, neurosyphilis, which is an infection of the brain or spinal cord

Diagnostic findings: Syphilis tests screen for and diagnose syphilis by looking for certain antibodies that are linked to the presence of Syphilis. The Centers for Disease Control and Prevention recommends that all pregnant people have a syphilis test at their first prenatal visit. Pregnant

people who are more likely to become infected with syphilis should be tested again at 28 weeks of pregnancy and at delivery.

The following methods can be employed:

1. First line; a. Rapid plasma reagin (RPR), which is a blood test.
b. Venereal Disease Research Laboratory (VDRL) test, which can be done on blood or spinal fluid

2. Second line of tests: This is necessary if the result of the first test is positive for antibodies linked to syphilis infections to confirm whether or not an individual has syphilis.

Usually, the second test looks for antibodies that the immune system makes only to fight off syphilis. If these antibodies are present, it means an individual has a syphilis infection now, or had a syphilis infection that was treated in the past. Common tests to check for syphilis antibodies include:

- a. Treponema pallidum particle agglutination assay (TP-PA)
- b. Fluorescent treponemal antibody absorption (FTA-ABS) test
- c. Microhemagglutination assay for antibodies to *T. pallidum* (MHA-TP)
- d. *T. pallidum* hemagglutination assay (TPHA)
- e. *T. pallidum* enzyme immunoassay (TP-EIA)
- f. Chemiluminescence immunoassays (CLIA)

Therapeutic Management:

Education

1. Educate patients on risk of spreading
 - a. Reproduction: Can pass from mother to child in utero
 - b. Sexuality: Passes between sexual partners
2. Patient Education
 - a. Incubation period varies: At maximum, takes 90 days to appear
 - b. Potential for multiple sexual partners or needle sharers during this window
 - c. Strongly encouraged to notify partners when diagnosed
 - d. High risk populations. c. Multiple sexual partners. d. IV drug users
3. High risk individuals should seek medical treatment with: New onset sores, Unexplainable rash

Medical Management

- a. Penicillin: Single dose given IM
- b. Doxycycline and Tetracycline Can be given if PCN allergy present
Cannot be given to pregnant women
- c. Ceftriaxone : Third line drug if first two options not viable

3. CHLAMYDIA

Aetiology

Chlamydia trachomatis (*C. trachomatis*) is the causative organism of Chlamydia. It's a very common sexually transmitted infection. Once a person is infected, they can spread chlamydia to their partners through intercourse, anal sex or oral sex. Infections can also occur when partners share sex toys that have become contaminated with the bacteria responsible for chlamydia.

There are lots of ways that the fluids from one person's genitals can transmit the bacteria that causes chlamydia.

Manual stimulation of the genitals or anus. Less commonly, infected vaginal fluid or semen can come in contact with a person's eye, causing an infection called conjunctivitis. For example, this can happen if you touch the genitals of an infected person and then rub your eyes without washing your hands first

Pathophysiology: The causative bacteria organism enters through sexual contact and reproduces within a host.

Clinical manifestations: Chlamydia bacteria often cause symptoms that are similar to cervicitis or a urinary tract infection (UTI). You may notice:

1. Female

- a. White, yellow or gray discharge from vagina that may be smelly (female).
- b. Pus in the urine (pyuria).
- c. Pain or a burning sensation during urination (dysuria).
- d. Bleeding in between periods (females).
- e. Painful periods.
- f. Painful intercourse (dyspareunia) (female).
- g. Itching or burning in and around the vagina (female).
- h. Dull pain in the lower part of your abdomen (female)

2. Male

Chlamydia bacteria most often infect your urethra, causing symptoms that are similar to nongonococcal urethritis. You may notice:

- a. Mucus-like or clear, watery discharge from the penis.
- b. Pain or a burning sensation during urination (dysuria).

Diagnostic findings: The Centers for Disease Control and Prevention recommends chlamydia screening for:

Sexually active women age 25 or younger. The rate of chlamydia infection is highest in this group, so a yearly screening test is recommended. Even if you've been tested in the past year, get tested when you have a new sex partner.

Pregnant women. You should be tested for chlamydia during your first prenatal exam. If you have a high risk of infection from changing sex partners or because your regular partner might be infected get tested again later in your pregnancy.

Women and men at high risk. People who have multiple sex partners, who don't always use a condom or men who have sex with men should consider frequent chlamydia screening. Other markers of high risk are current infection with another sexually transmitted infection and possible exposure to an STI through an infected partner.

a. **Urine test:** A sample of urine is analyzed in the laboratory for presence of this infection.

b. **Swab:** For women, a swab of the discharge from the cervix for culture or antigen testing for chlamydia. This can be done during a routine Pap test. Some women prefer to swab their vaginas themselves, which has been shown to be as diagnostic as doctor-obtained swabs.

For men, the doctor inserts a slim swab into the end of the penis to get a sample from the urethra. In some cases, the doctor will swab the anus.

CHAPTER ELEVEN:

SEXUALLY ASSOCIATED DISEASES 2: VIRAL INFECTIONS

1. Human papilloma virus

Aetiology/Epidemiology: Human papilloma virus (HPV) is the name given to certain categories of viruses, which are very common. These viruses do not normally cause health break down in most people, except some that are capable of causing genital warts, and sometimes cancer.

It is a skin condition, hence it affects the skin. Research has shown that HPV has more than 100 types of HPV in our world today.

Genital warts usually appear as a small bump or group of bumps in the genital area. They can be small or large, raised or flat, or shaped like a cauliflower.

HPV is the most common STI. There were about 43 million HPV infections in 2018, many among people in their late teens and early 20s. According to a trusted source, Human papilloma virus (HPV) is the most common sexually transmitted infection (STI) in the United States.

Pathophysiology: The HPV virus is a "small, non-enveloped, double stranded DNA virus that infects the mucosal or cutaneous epithelium" (Valentino & Poronsky, 2015, p. 156).

- Since HPV affects epithelial cells and does not enter the bloodstream, "having an HPV infection in one part of the body should not cause infection in another part (McCance & Huether, 2014, p. 424).
- Once HPV gets into the epithelial cell, "the virus begins to make proteins that can interfere with normal functions in the cell, enabling the cell to grow in an uncontrolled manner and to avoid apoptosis" (McCance & Huether, 2014, p. 424). Underlying Pathophysiology

- HPV modifies the DNA damage response (DDR) “pathways by interacting with many proteins, including ATM, ATR, MRN, γ -H2AX, Chk1, Chk2, p53, BRCA1, BRCA2, RAD51...”(Low et al, 2016, p. 28). and a few others.
- The HPV virus “can activate and dysregulate DDR pathways throughout various stages of their life cycles to replicate itself in host cells” (Low et al, 2016, p. 29).
- Cell biology during a different periods of a woman’s life can make her more susceptible to contracting the virus (Choma & McKeever, 2015, p.51).

Clinical manifestations:

Plantar warts: Plantar warts are hard, grainy growths that usually appear on the heels or balls of your feet. These warts might cause discomfort.

Common warts: Common warts appear as rough, raised bumps and usually occur on the hands and fingers. In most cases, common warts are simply unsightly, but they can also be painful or susceptible to injury or bleeding.

Genital warts: These appear as flat lesions, small cauliflower-like bumps or tiny stemlike protrusions. In women, genital warts appear mostly on the vulva but can also occur near the anus, on the cervix or in the vagina.

In men, genital warts appear on the penis and scrotum or around the anus. Genital warts rarely cause discomfort or pain, though they may itch or feel tender.

Flat warts: Flat warts are flat-topped, slightly raised lesions. They can appear anywhere, but children usually get them on the face and men tend to get them in the beard area. Women tend to get them on the legs.

Diagnostic findings: warts are generally visible, but if they aren't visible, these tests can be conducted:

1. DNA test: This test, conducted on cells from the cervix, can recognize the DNA of the high-risk varieties of HPV that have been linked to genital cancers. It's recommended for women 30 and older in addition to the Pap test.

2. Vinegar (acetic acid) solution test: A vinegar solution applied to HPV-infected genital areas turns them white. This may help in identifying difficult-to-see flat lesions.
3. Pap test: A sample of cells are collected from the cervix or vagina to send for laboratory analysis. Pap tests can reveal abnormalities that can lead to cancer.

Therapeutic Management:

Warts often go away without treatment. This is true particularly in children.

Medications

Medications to eliminate warts are generally topical, hence it's applied on the lesions. It can take several application of these medications before the lesions can be eliminated.

1. Trichloroacetic acid: it's a chemical that burns off warts on the palms, soles and genitals. It might cause local irritation.
2. Salicylic acid: salicylic acid work by removing layers of a wart a little at a time. For use on common warts, salicylic acid can cause skin irritation and isn't for use on your face.
3. Imiquimod: This prescription cream might enhance your immune system's ability to fight HPV. Common side effects include redness and swelling at the application site.
4. Podofilox: This works by destroying genital wart tissue. Podofilox may cause burning and itching where it's applied.

Surgical Intervention

The second line of treatment when medications do not work is surgery and other methods. The surgery is aimed at removing warts.

The exams of surgical procedures and others are:

1. Freezing with liquid nitrogen (cryotherapy)
2. Burning with an electrical current (electrocautery)
3. Laser surgery

2. GENITAL HERPS

Aetiology/Epidemiology: Genital herpes is one of the Infections caused by sexually transmitted Diseases. There are two types of the herpes simplex virus (HSV) that causes genital herpes:

HSV-1. This type usually causes cold sores, but it can also cause genital herpes.

HSV-2. This type usually causes genital herpes, but it can also cause cold sores.

The World Health Organization stated that in 2016, about 3.7 billion Trusted Source people under age 50 years had contracted HSV-1. In the same year, around 491 million people ages 15 to 49 years had an HSV-2 infection.

Although genital herpes is typically caused by HSV-2, the infection can also be caused by HSV-1.

According to the WHO's latest available statistics, it was estimated that 491.5 million people had an HSV-2 infection in 2016. This is over one-tenth of the world's population ages 15 to 49 years.

The WHO also estimates that 3.7 billion people had an HSV-1 infection in the same year, which accounts for around two-thirds of the world's population under age 50 years.

Pathophysiology: The viruses enter the body through skin abrasions or mucous membranes (the thin layers of tissue that line the openings of the body, found in nose, mouth, and genitals).

Once the viruses are inside the body, they incorporate themselves into the cells. Viruses tend to multiply or adapt to their environments very easily, which makes treating them difficult. The virus causes herpetic sores, which are painful blisters (fluid-filled bumps) that can break open and ooze fluid.

HSV-1 or HSV-2 can be found in bodily fluids, including: saliva, semen, and vaginal secretions

Clinical manifestations: How do you recognize the symptoms of genital herpes?

The appearance of blisters is known as an outbreak. On average, a first outbreak will appear 4 days after contracting the virus, according to the

Centers for Disease Control and Prevention (CDC). However, it can take as little as 2 days, or as much as 12 days or more, to appear.

General symptoms for those with a penis include blisters on the: penis, scrotum, buttocks (near or around the anus).

Blisters around or near the: vagina, anus, buttocks.

General symptoms may include: Blisters may appear in the mouth and on the lips, face, and anywhere else that came into contact with areas of infection, The area that has contracted the condition often starts to itch, or tingle, before blisters actually appear, The blisters may become ulcerated (open sores) and ooze fluid, A crust may appear over the sores within a week of the outbreak, The lymph glands may become swollen. Lymph glands fight infection and inflammation in the body, the viral infection may cause headaches, body aches, and fever.

Babies who are born with genital herpes can develop very severe complications and experience: blindness, brain damage, death.

Diagnosing genital herpes: Genital herpes can be typically diagnosed by the transmission of a visual examination of the herpes sores. Laboratory tests are not necessarily recommended. Though a blood test can diagnose HSV before an outbreak occurs. However, if there has not been exposure to the virus and there are no symptoms being displayed, it's not always necessary to be screened for HSV-1 or HSV-2.

Therapeutic Management

The management of this disease is targeted towards reducing the outbreak, but can't be cured.

Medications

Antiviral drugs may help speed up the healing time for sores and reduce pain. Medications may be taken at the first signs of an outbreak (tingling, itching, and other symptoms) to help reduce the symptoms.

3. HIV/ AIDS

Aetiology: HIV (human immunodeficiency virus) is a virus that attacks cells that help the body fight infection, making a person more vulnerable to other infections and diseases. It is spread by contact with certain bodily fluids of a person with HIV, most commonly during

unprotected sex (sex without a condom or HIV medicine to prevent or treat HIV), or through sharing injection drug equipment.

The late stage of HIV, when the body's immune system is badly damaged emanate into AIDS.

In the U.S., most people with HIV do not develop AIDS because taking HIV medicine as prescribed stops the progression of the disease.

A person with HIV is considered to have progressed to AIDS when: the number of their CD4 cells falls below 200 cells per cubic millimeter of blood (200 cells/mm³). (In someone with a healthy immune system, CD4 counts are between 500 and 1,600 cells/mm³.) OR they develop one or more opportunistic infections regardless of their CD4 count.

Clinical manifestations: **Within** a few weeks of HIV infection, flu-like symptoms such as fever, sore throat and fatigue can occur. Then the disease is usually asymptomatic until it progresses to AIDS. AIDS symptoms include weight loss, fever or night sweats, fatigue and recurrent infections.

Diagnostic findings:

Antibody tests: This look for antibodies to HIV in a person's blood or oral fluid. Antibody tests can take 23 to 90 days to detect HIV after exposure. Antigen/antibody tests look for both HIV antibodies and antigens.

Therapeutic Management

Luckily, however, effective treatment with HIV medicine (called antiretroviral therapy or ART) is available. If taken as prescribed, HIV medicine can reduce the amount of HIV in the blood (also called the viral load) to a very low level. This is called viral suppression. If a person's viral load is so low that a standard lab can't detect it, this is called having an undetectable viral load. People with HIV who take HIV medicine as prescribed and get and keep an undetectable viral load can live long and healthy lives and will not transmit HIV to their HIV-negative partners through sex.

In addition, there are effective methods to prevent getting HIV through sex or drug use, including pre-exposure prophylaxis (PrEP), medicine people at risk for HIV take to prevent getting HIV from sex or injection drug use, and post-exposure prophylaxis (PEP), HIV medicine taken

within 72 hours after a possible exposure to prevent the virus from taking hold. Learn about other ways to prevent getting or transmitting HIV.

CHAPTER TWELVE

**SEXUALLY ASSOCIATED
DISEASES 3:**

12. 0. PARASITIC INFECTIONS

12.1. Trichomoniasis

Aetiology/Epidemiology: The most common parasitic sexually transmitted infection is Trichomoniasis.

It result from a Parasite called Trichomonas vaginalis.

Trichomoniasis can affect all genders. Women (especially older women) are more likely than men to get the disease. Black women are more likely to get this disease.

Trichomoniasis is the most common curable STD affecting both men and women in America. Approximately 3.7 million people have the disease.

Clinical manifestations: This disease affect large number of people, hence spreads so easily. About 70% of people with this disease do not show symptoms. Men rarely show any signs of infection. When symptoms occur, they tend to appear within five to 28 days after exposure. The individual may experience: Thin (or sometimes foamy) white, yellow or greenish vaginal discharge that has a bad odor, White discharge from the penis, Genital itching or irritation, Burning or painful urination, Burning after ejaculation, Pain or discomfort during intercourse.

Diagnostic findings: a. Physical exam: For women, this exam includes a pelvic exam.

b. Lab test: a sample of the genital discharge is collected and examined under a microscope to check for signs of infection. The vaginal swab

collected may be sent to the lab for further testing if trichomonads are not seen under the microscope.

Therapeutic Management: A parasite called *Trichomonas vaginalis* causes this STD. Once a person is infected, the disease can be transmitted to someone else through:

Vaginal-penile or vaginal-vaginal intercourse, Anal sex, Oral sex.

Without treatment, trich can last for months or even years. It doesn't go away on its own. The entire time you're infected, you can give the STD to your sexual partners.

Oral anti-infective medications kill trich. The doctor may prescribe metronidazole (Flagyl) or tinidazole (Tindamax). It's important to keep the following points in mind while undergoing treatment: A single medication dose cures up to 95% of infected women. Men and women may need to take the medication for five to seven days.

Both sexual partners must be treated for trich, otherwise, the infection can be passed continuously.

CHAPTER THIRTEEN

**GENERAL DISEASE
PREVENTION 1:
IMMUNOLOGICAL CONCEPTS**

13. 0. Definition;

Immunology is the scientific study of the body's resistance to invasion by other organisms (i.e., immunity). In a medical sense, immunology deals with the body's system of defense against disease-causing microorganisms and with disorders in that system's functioning. The artificial induction of immunity against disease has been known in the West at least since Edward Jenner used cowpox injections to protect people from smallpox in 1796. But the scientific basis for immunology was not established until a century later, when it was recognized that:

(1) Proliferating microorganisms in the body cause many infectious diseases and (2) the body has certain chemical and cellular components that recognize and destroy foreign substances (antigens) within the body. This new understanding led to highly successful techniques of immunization that could mobilize and stimulate the body's natural defenses against infectious disease.

13. 0.1. ORIGIN AND BENEFITS

It was only in the 20th century, however, that a comprehensive understanding was gained of the formation, mobilization, action, and interaction of antibodies and antigen-reactive lymphocytes, which are the two main active elements of the immune system. Modern immunology, besides using such basic techniques as vaccination, has become increasingly selective and sophisticated in its manipulation of the body's immune system through drugs and other agents in efforts to

achieve a desired therapeutic goal. Immunologic understanding is crucial to the treatment of allergies, which are themselves hypersensitive reactions by the body's immune system to the presence of harmless antigens such as pollen grains. Immunosuppressive techniques use drugs to suppress the immune system's tendency to reject and attack antigenic bone grafts and organ transplants that have been medically introduced into the host tissue. Immunology also encompasses the increasingly important study of autoimmune diseases, in which the body's immune system attacks some constituent of its own tissues as if it were a foreign body. The study of immune deficiencies has become an area of intensive research since the appearance of AIDS (acquired immune deficiency syndrome), a disease that destroys the body's immune system and for which there is currently no cure.

13.0.2. History

Dramatic though they undoubtedly were, the advances in chemotherapy still left one important area vulnerable, that of the viruses. It was in bringing viruses under control that advances in immunology—the study of immunity—played such a striking part. One of the paradoxes of medicine is that the first large-scale immunization against a viral disease was instituted and established long before viruses were discovered. When Edward Jenner introduced vaccination against the virus that causes smallpox, the identification of viruses was still 100 years in the future. It took almost another half century to discover an effective method of producing antiviral vaccines that were both safe and effective.

13. 2. HUMAN BODY DEFENSE MECHANISM

Our body also, fights against foreign agents thorough a network of cells and bodily substances that detect and combat micro-organisms. The process by which the body does this, is what I called body defence mechanism. We shall discuss it in details.

The immune system is made up of special organs, cells and chemicals that fight infection (microbes).

The immune system is a complex network of cells and proteins that defends the body against infection.

The immune system keeps a record of every germ (microbe) it has ever defeated so it can recognise and destroy the microbe quickly if it enters the body again.

The immune system keeps a record of every microbe it has ever defeated, in types of white blood cells (B- and T-lymphocytes) known as memory cells. This means it can recognise and destroy the microbe quickly if it enters the body again, before it can multiply and make you feel sick.

Some infections, like the flu and the common cold, have to be fought many times because so many different viruses or strains of the same type of virus can cause these illnesses. Catching a cold or flu from one virus does not give you immunity against the others.

For example, a rise in body temperature, or fever, can happen with some infections. This is actually an immune system response. A rise in temperature can kill some microbes. Fever also triggers the body's repair process.

13.3. HUMAN DEFENSE SYSTEM

The process of the body's defense mechanism is facilitated through certain bodily parts, which are the main parts of the immune system.

These parts are the bone marrow, the thymus, the spleen, the antibodies, the complement system, the white blood cells, and the lymphatic system.

Let us discuss them in details.

13.4. PARTS OF THE DEFENSE SYSTEM

- **White blood cells-** White blood cells include lymphocytes (such as B-cells, T-cells and natural killer cells), and many other types of immune cells.

White blood cells are the key players in the immune system. They are made in the bone marrow and are part of the lymphatic system.

White blood cells move through blood and tissue throughout your body, looking for foreign invaders (microbes) such as bacteria, viruses,

parasites and fungi. When they find them, they launch an immune attack.

- **Lymphatic System-** The lymphatic system is a network of delicate tubes throughout the body. The main roles of the lymphatic system are to: manage the fluid levels in the body react to bacteria, deal with cancer cells, deal with cell products that otherwise would result in disease or disorders, absorb some of the fats in our diet from the intestine.

The lymphatic system is made up of:

lymph nodes (also called lymph glands) -- which trap microbes
 lymph vessels -- tubes that carry lymph, the colourless fluid that bathes your body's tissues and contains infection-fighting white blood cells and white blood cells (lymphocytes).

- **Bone marrow-** Bone marrow is the spongy tissue found inside your bones. It produces the red blood cells our bodies need to carry oxygen, the white blood cells we use to fight infection, and the platelets we need to help our blood clot.
- **Complement system-**The complement system is made up of proteins whose actions complement the work done by antibodies.
- **Lymphatic system-**Thymus -The thymus filters and monitors your blood content. It produces the white blood cells called T-lymphocytes.
- **Spleen-** The spleen is a blood-filtering organ that removes microbes and destroys old or damaged red blood cells. It also makes disease-fighting components of the immune system (including antibodies and lymphocytes).
- **Antibodies-**Antibodies help the body to fight microbes or the toxins (poisons) they produce. They do this by recognising substances called antigens on the surface of the microbe, or in the chemicals they produce, which mark the microbe or toxin as being foreign. The antibodies then mark these antigens for destruction. There are many cells, proteins and chemicals involved in this attack.

13.5. BASIC PROBLEMS OF THE IMMUNE SYSTEM

The problem commonly experienced in the immune system could be either an over- or underactive immune system.

What is overactivity of the immune system? This can take many forms, including:

Allergic diseases - where the immune system makes an overly strong response to allergens. Allergic diseases are very common. They include allergies to foods, medications or stinging insects, anaphylaxis (life-threatening allergy), hay fever (allergic rhinitis), sinus disease, asthma, hives (urticaria), dermatitis and eczema.

Autoimmune diseases - where the immune system mounts a response against normal components of the body. Autoimmune diseases range from common to rare. They include multiple sclerosis, autoimmune thyroid disease, type 1 diabetes, systemic lupus erythematosus, rheumatoid arthritis and systemic vasculitis.

What is Underactivity of the immune system? It is also called immunodeficiency, can:

Be inherited - examples of these conditions include primary immunodeficiency diseases such as common variable immunodeficiency (CVID), x-linked severe combined immunodeficiency (SCID) and complement deficiencies.

Arise as a result of medical treatment - this can occur due to medications such as corticosteroids or chemotherapy.

Be caused by another disease - such as HIV/AIDS or certain types of cancer.

An underactive immune system does not function correctly and makes people vulnerable to infections. It can be life threatening in severe cases, which includes:

-People who have had an organ transplant need immunosuppression treatment to prevent the body from attacking the transplanted organ.

Immunoglobulin therapy -Immunoglobulins (commonly known as antibodies) are used to treat people who are unable to make enough of their own, or whose antibodies do not work properly. This treatment is known as immunoglobulin therapy.

Until recently, immunoglobulin therapy in Australia mostly involved delivery of immunoglobulins through a drip into the vein – known as intravenous immunoglobulin (IVIg) therapy. Now, subcutaneous immunoglobulin (SCIg) can be delivered into the fatty tissue under the skin, which may offer benefits for some patients. This is known as subcutaneous infusion or SCIg therapy.

Subcutaneous immunoglobulin is similar to intravenous immunoglobulin. It is made from plasma – the liquid part of blood containing important proteins like antibodies.

13.6. BASIC WORKING FUNCTION OF IMMUNIZATION

Immunisation works by copying the body's natural immune response. A vaccine (a small amount of a specially treated virus, bacterium or toxin) is injected into the body. The body then makes antibodies to it.

If a vaccinated person is exposed to the actual virus, bacterium or toxin, they won't get sick because their body will recognise it and know how to attack it successfully. Vaccinations are available against many diseases, including measles and tetanus.

The immunisations you may need are decided by your health, age, lifestyle and occupation. Together, these factors are referred to as HALO, which is defined as:

Health - some health conditions or factors may make you more vulnerable to vaccine-preventable diseases. For example, premature birth, asthma, diabetes, heart, lung, spleen or kidney conditions, Down syndrome and HIV will mean you may benefit from additional or more frequent immunisations

Age - at different ages you need protection from different vaccine-preventable diseases. National Immunisation Program sets out recommended immunisations for babies, children, older people and other people at risk.

Lifestyle - lifestyle choices can have an impact on your immunisation needs. Travelling overseas to certain places, planning a family, sexual activity, smoking, and playing contact sport that may expose you directly to someone else's blood, will mean you may benefit from additional or more frequent immunizations.

occupation - you are likely to need extra immunisations, or need to have them more often, if you work in an occupation that exposes you to vaccine-preventable diseases or puts you into contact with people who are more susceptible to problems from vaccine-preventable diseases (such as babies or young children, pregnant women, the elderly, and people with chronic or acute health conditions). For example, if you work in aged care, childcare, healthcare, emergency services or sewerage repair and maintenance, discuss your immunisation needs with your doctor. Some employers help with the cost of relevant vaccinations for their employees.

13.7. SUPPLEMENTARY BODY DEFENSE

It is not only the immune system that defends the body, the body has several other ways to defend itself against microbes, including:

Skin - a waterproof barrier that secretes oil with bacteria-killing properties.

Lungs - mucous in the lungs (phlegm) traps foreign particles, and small hairs (cilia) wave the mucous upwards so it can be coughed out.

Digestive tract - the mucous lining contains antibodies, and the acid in the stomach can kill most microbes.

Body fluids- like skin oil, saliva and tears contain anti-bacterial enzymes that help reduce the risk of infection. The constant flushing of the urinary tract and the bowel also helps.

CHAPTER FOURTEEN

**GENERAL DISEASE
PREVENTION 2: VACCINES,
DERIVATIVES AND USES**

14. 0. Vaccine:

A suspension of weakened, killed, or fragmented microorganisms or toxins or other biological preparation, such as those consisting of antibodies, lymphocytes, or messenger RNA (mRNA), that is administered primarily to prevent disease.

A vaccine can confer active immunity against a specific harmful agent by stimulating the immune system to attack the agent. Once stimulated by a vaccine, the antibody-producing cells, called B cells (or B lymphocytes), remain sensitized and ready to respond to the agent should it ever gain entry to the body. A vaccine may also confer passive immunity by providing antibodies or lymphocytes already made by an animal or human donor. Vaccines are usually administered by injection (parenteral administration), but some are given orally or even nasally (in the case of flu vaccine). Vaccines applied to mucosal surfaces, such as those lining the gut or nasal passages, seem to stimulate a greater antibody response and may be the most effective route of administration. (For further information, see immunization.)

14.1. The first vaccines:

The first vaccine was introduced by British physician Edward Jenner, who in 1796 used the cowpox virus (vaccinia) to confer protection against smallpox, a related virus, in humans. Prior to that use, however, the principle of vaccination was applied by Asian physicians who gave children dried crusts from the lesions of people suffering from smallpox to protect against the disease. While some developed immunity, others

developed the disease. Jenner's contribution was to use a substance similar to, but safer than, smallpox to confer immunity. He thus exploited the relatively rare situation in which immunity to one virus confers protection against another viral disease. In 1881 French microbiologist Louis Pasteur demonstrated immunization against anthrax by injecting sheep with a preparation containing attenuated forms of the bacillus that causes the disease. Four years later he developed a protective suspension against rabies.

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14.2. Vaccine types

The challenge in vaccine development consists in devising a vaccine strong enough to ward off infection without making the individual seriously ill. To that end, researchers have devised different types of vaccines. Weakened, or attenuated, vaccines consist of microorganisms that have lost the ability to cause serious illness but retain the ability to stimulate immunity. They may produce a mild or subclinical form of the disease. Attenuated vaccines include those for measles, mumps, polio (the Sabin vaccine), rubella, and tuberculosis. Inactivated vaccines are those that contain organisms that have been killed or inactivated with heat or chemicals. Inactivated vaccines elicit an immune response,

but the response often is less complete than with attenuated vaccines. Because inactivated vaccines are not as effective at fighting infection as those made from attenuated microorganisms, greater quantities of inactivated vaccines are administered. Vaccines against rabies, polio (the Salk vaccine), some forms of influenza, and cholera are made from inactivated microorganisms. Another type of vaccine is a subunit vaccine, which is made from proteins found on the surface of infectious agents. Vaccines for influenza and hepatitis B are of that type. When toxins, the metabolic by-products of infectious organisms, are inactivated to form toxoids, they can be used to stimulate immunity against tetanus, diphtheria, and whooping cough (pertussis).

In the late 20th century, advances in laboratory techniques allowed approaches to vaccine development to be refined. Medical researchers could identify the genes of a pathogen (disease-causing microorganism) that encode the protein or proteins that stimulate the immune response to that organism. That allowed the immunity-stimulating proteins (called antigens) to be mass-produced and used in vaccines. It also made it possible to alter pathogens genetically and produce weakened strains of viruses. In that way, harmful proteins from pathogens can be deleted or modified, thus providing a safer and more-effective method by which to manufacture attenuated vaccines.

Recombinant DNA technology has also proven useful in developing vaccines to viruses that cannot be grown successfully or that are inherently dangerous. Genetic material that codes for a desired antigen is inserted into the attenuated form of a large virus, such as the vaccinia virus, which carries the foreign genes “piggyback.” The altered virus is injected into an individual to stimulate antibody production to the foreign proteins and thus confer immunity. The approach potentially enables the vaccinia virus to function as a live vaccine against several diseases, once it has received genes derived from the relevant disease-causing microorganisms. A similar procedure can be followed using a modified bacterium, such as *Salmonella typhimurium*, as the carrier of a foreign gene.

Vaccines against human papillomavirus (HPV) are made from viruslike particles (VLPs), which are prepared via recombinant technology. The vaccines do not contain live HPV biological or genetic material and therefore are incapable of causing infection. Two types of HPV vaccines have been developed, including a bivalent HPV vaccine, made using VLPs of HPV types 16 and 18, and a tetravalent vaccine, made with VLPs of HPV types 6, 11, 16, and 18.

Another approach, called naked DNA therapy, involves injecting DNA that encodes a foreign protein into muscle cells. The cells produce the foreign antigen, which stimulates an immune response.

Vaccines based on RNA have been of particular interest as a means of preventing diseases such as influenza, cytomegalovirus infection, and rabies. Messenger RNA (mRNA) vaccines are advantageous because the way in which they are made allows them to be developed more quickly than vaccines made via other methods. In addition, their production can be standardized, enabling rapid scale-up for the manufacture of large quantities of vaccine. Novel mRNA vaccines are safe and effective; they do not contain live virus, nor does the RNA interact with human DNA.

14.3. FORMS OF VACCINES

1. LIVE VACCINES

The first live vaccine was cowpox virus introduced by Edward Jenner as a vaccine for smallpox, however; variolation (innoculation using pus from a patient with a mild case of smallpox) has been in use for over a thousand years. Live vaccines are used against a number of viral infections (polio and Sabin vaccine), measles, mumps, rubella, chicken pox, hepatitis A, yellow fever, etc.)

The only example of live bacterial vaccine is one against tuberculosis (*Mycobacterium bovis*: Bacille Calmette-Guerin vaccine: BCG). This is used in many African, European and Asian countries but not in the United States. Whereas many studies have shown the efficacy of BCG

vaccine, a number of studies also cast doubt on its benefits. Live vaccines normally produce self-limiting non-clinical infections and lead to subsequent immunity, both humoral and cell-mediated, the latter being essential for intracellular pathogens. However, they carry a serious risk of causing overt disease in immune compromised individuals. Furthermore, since live vaccines are often attenuated (made less pathogenic) by passage in animals or thermal mutation, they can revert to their pathogenic form and cause serious illness. It is for this reason that

103 live polio (Sabin) vaccine, which was used for many years, has been replaced in many countries by the inactivated (Salk) vaccine.

2. KILLED VACCINES

Killed (heat, chemical or UV irradiation) viral vaccines include those for polio (Salk vaccine), influenza, rabies, influenza, rabies, etc. Most bacterial vaccines are killed organisms (typhoid, cholera, plague, pertussis, etc.)

3. ADJUVANTS

Weaker antigens may be rendered more immunogenic by the addition of other chemicals. Such chemicals are known as adjuvants. There are many biological and chemical substances that have been used in experimental conditions. However, only aluminum salts (alum) are approved for human use and it is incorporated in DTP vaccine. Furthermore, pertussis itself has adjuvant effects. Adjuvants used experimentally include mixtures of oil and detergents, with (Freund's complete adjuvant) or without (Freund's incomplete adjuvant) certain bacteria. Bacteria most often used in an adjuvant are Mycobacteria (BCG) and Nocardia. In some instances, sub-cellular fractions of these bacteria can also be used effectively as adjuvants. Newer adjuvant formulations include synthetic polymers and oligonucleotides. Most adjuvants recognize TOLL-like receptors, thus activating mononuclear phagocytes and inducing selective cytokines that can enhance Th1 or Th2 responses, depending on the nature of the adjuvant.

The protective immunity conferred by a vaccine may be life-long (measles, mumps, rubella, small pox, tuberculosis, yellow fever, etc.) or may last as little as a few months (cholera). The primary immunization may be given at the age of 2 to 3 months (diphtheria, pertussis, tetanus, polio), or 13 to 15 months (mumps, measles, rubella). The currently recommended schedule for routine immunization in the United States (recommended by CDC and AIP). This schedule is revised on a yearly basis or as need by the CDC Advisory Committee on Immunization Practice (AICP).

14.4. PROPHYLACTIC VERSUS THERAPEUTIC IMMUNIZATION

Most vaccines are given for prophylaxis, i.e. prior to exposure to the pathogen. However, some vaccines can be administered therapeutically, i.e. post exposure (e.g. rabies virus). The effectiveness of this mode of immunization depends on the rate of replication of the pathogen, incubation period and the pathogenic mechanism. For this reason, only a booster shot with tetanus is sufficient if the exposure to the pathogen is within less than 10 years and if the exposure is minimal (wounds are relative superficial).

In a situation where pathogen has a short incubation period, the pathogenic mechanism is such that only a small amount of pathogenic molecules could be fatal (e.g., tetanus and diphtheria) and/or a bolus of infection is relatively large, both passive and active post exposure immunization are essential. Passive prophylactic immunization is also normal in cases of defects in the immune system, such as hypogammaglobulinemias.

14.5. BENEFITS OF VACCINATION

In addition to the development of memory B cells, which are capable of triggering a secondary immune response upon exposure to the pathogen targeted by a vaccine, vaccination is also beneficial at the population level. When a sufficient number of individuals in a population are immune to a disease, as would occur if a large proportion of a

population were vaccinated, herd immunity is achieved. That means that if there is random mixing of individuals within the population, then the pathogen cannot be spread throughout the population. Herd immunity acts by breaking the transmission of infection or by lessening the chances of susceptible individuals coming in contact with a person who is infectious. Herd immunity provides a measure of protection to individuals who are not personally immune to the disease—for instance, individuals who, because of their age or underlying medical conditions, cannot receive vaccines or individuals who received vaccines but remain susceptible. Herd immunity played an important role in the successful eradication of smallpox, and it is vital in preventing the spread of diseases such as polio and measles.

14.6. Adverse reactions

Vaccination carries some risk of reaction, though adverse effects typically are very rare and very mild. The most common reactions to vaccines include redness and soreness around the vaccination site. More severe adverse reactions, such as vomiting, high fever, seizure, brain damage, or death, are possible for some vaccines. Such reactions are exceptionally rare, however—occurring in less than one in a million people for most vaccines. Severe reactions also tend to affect only certain populations, such as persons whose immune systems are compromised by preexisting disease (e.g., HIV/AIDS) or who are undergoing chemotherapy. Claims have been made that vaccines are responsible for certain adverse health conditions, particularly autism, speech disorders, and inflammatory bowel disease. Some of those claims focused on thimerosal, a mercury-containing compound used as a preservative in vaccines. Some people believed that autism was a form of mercury poisoning, caused specifically by thimerosal in childhood vaccines. Those claims have been discredited. Still, misinformation and fear generated by false claims about associations between autism and vaccines had a significant impact on individuals' perceptions about vaccine safety. In addition, most individuals in countries where vaccination is widespread have never personally experienced vaccine-preventable disease. Thus, the focus of concern for

some people shifted from the negative effects of vaccine-preventable disease to the possible negative effects of the vaccines themselves.

CHAPTER FIFTEEN

GENERAL DISEASE PREVENTION 3: SANITATION

15.0. introduction

For every individual, it is important to lead a healthy life. The 7th of April has been declared as “World Health Day by” the World Health Organization (WHO) in view of creating awareness about the importance of health. To stay healthy, it is important to understand a few concept surrounding public health and sanitation (hygiene).The WHO defines health as – “a complete state of physical, mental and social well-being, and not merely the absence of disease or infirmity.” That is to say, a person cannot be termed healthy merely by being in a disease-free state. Physical and mental health are equally important and require a hygienic condition. Health and hygiene are two terms that are correlated.

Hygiene can be outlined as the practice of a few habits in order to maintain good health, overall. Maintenance of hygiene can be at the community level (social hygiene) or personal level (personal hygiene).

Personal hygiene: It covers physical exercise, cleanliness, sleep, proper rest and other related practices such as keeping away from consuming alcohol, smoking, drugs etc. Many diseases can be prevented to a great extent just by maintaining good personal hygiene.

Social hygiene: The surrounding and other public places around us are our society. An individual’s mental and physical condition is greatly influenced by a good environment. Improper, unkempt and untidy surroundings and inappropriate ways of waste disposal in public places

results in the unhealthy surrounding. Such practices can cause an alarming growth of rodents, pathogens and other microbes, which can make us unwell. Consequently, both social and personal hygiene are vital aspects.

What is Sanitation?

Sanitation refers to public health conditions such as drinking clean water, sewage treatment, etc. All the effective tools and actions that help in keeping the environment clean come under sanitation.

Sanitation refers to public health conditions related to clean drinking water and treatment and disposal of human excreta and sewage. Proper sanitation promotes health, improves the quality of the environment and thus, the quality of life in a community. Sanitation refers to the safe collection, transportation, treatment and disposal of human wastes. In developing countries, improvements in practices of disposing of human excreta are crucial to raising levels of public health. Preventing human contact with feces is part of sanitation, as is hand washing with soap. Sanitation systems aim to protect human health by providing a clean environment that will stop the transmission of disease, especially through the fecal–oral route. There are many diseases which are easily transmitted in communities that have low levels of sanitation. They include: diarrhea, ascariasis (a type of intestinal worm infection or helminthiasis), cholera, hepatitis, polio, schistosomiasis, and trachoma, to name just a few. An increasing amount of literature suggests that health problems result from the lack of sanitation facilities, especially among the urban poor living in overcrowded informal settlements. Invariably, it is the poor who suffer the most from the absence of safe water and sanitation because they lack not only the means to provide such facilities but also the information on how to minimize the ill-effects of the unsanitary conditions in which they live. As a result, the negative effects of unsanitary living conditions lower the productive potential of the people who can least afford it. Proper sanitation facilities (for example, toilets and latrines) promote health because they allow people to dispose of their waste appropriately, preventing

contamination of their environment and reducing risk to themselves and their neighbors. Throughout the world, many people do not have access to sanitation facilities, resulting in improper waste disposal that safely contain waste away from human contact and ensure that waste is properly treated prior to environmental discharge and other risks.

Furthermore; Sanitation refers to public health conditions related to clean drinking water and treatment and disposal of human excreta and sewage. Preventing human contact with feces is part of sanitation, as is hand washing with soap. Sanitation systems aim to protect human health by providing a clean environment that will stop the transmission of disease, especially through the fecal–oral route. example, diarrhea, a main cause of malnutrition and stunted growth in children, can be reduced through adequate sanitation. There are many other diseases which are easily transmitted in communities that have low levels of sanitation, such as ascariasis (a type of intestinal worm infection or helminthiasis), cholera, hepatitis, polio, schistosomiasis, and trachoma, to name just a few.

15.1. TYPES OF SANITATION

The sanitation system: collection, transport, treatment, disposal or reuse.

A range of sanitation technologies and approaches exists. Some examples are community-led total sanitation, container-based sanitation, ecological sanitation, emergency sanitation, environmental sanitation, onsite sanitation and sustainable sanitation. A sanitation system includes the capture, storage, transport, treatment and disposal or reuse of human excreta and wastewater. Activities within the sanitation system may focus on the nutrients, water, energy or organic matter contained in excreta and wastewater. This is referred to as the "sanitation value chain" or "sanitation economy". The people responsible for cleaning, maintaining, operating, or emptying a sanitation technology at any step of the sanitation chain are called "sanitation workers".

Several sanitation "levels" are being used to compare sanitation service levels within countries or across countries. sanitation ladder defined by the Joint Monitoring Programme in 2016 starts at open defecation and moves upwards using the terms "unimproved", "limited", "basic", with the highest level being "safely managed". is particularly applicable to developing countries.

The Human Right to Water and Sanitation was recognized by the United Nations (UN) General Assembly in 2010. Sanitation is a global development priority and the subject of Sustainable Development Goal 6. The estimate in 2017 by JMP states that 4.5 billion people currently do not have safely managed sanitation. Lack of access to sanitation has an impact not only on public health but also on human dignity and personal safety. The term sanitation is connected with various descriptors or adjectives to signify certain types of sanitation systems (which may deal only with human excreta management or with the entire sanitation system, i.e. also greywater, stormwater and solid waste management) – in alphabetical order:

Basic sanitation

In 2017, JMP defined a new term: "basic sanitation service". This is defined as the use of improved sanitation facilities that are not shared with other households. A lower level of service is now called "limited sanitation service" which refers to use of improved sanitation facilities that are shared between two or more households.

Container-based sanitation

Container-based sanitation (abbreviated as CBS) refers to a sanitation system where toilets collect human excreta in sealable, removable containers (also called cartridges) that are transported to treatment facilities. type of sanitation involves a commercial service which provides certain types of portable toilets, and delivers empty containers when picking up full ones. The service transports and safely disposes of or reuses collected excreta. The cost of collection of excreta is usually borne by the users. With suitable development, support and functioning partnerships, CBS can be used to provide low-income urban

populations with safe collection, transport and treatment of excrement at a lower cost than installing and maintaining sewers.[28] In most cases, CBS is based on the use of urine-diverting dry toilets.

Community-based sanitation

Community-based sanitation is related to decentralized wastewater treatment (DEWATS). **Community-led total sanitation**

Community-led total sanitation (CLTS) is an approach used mainly in developing countries to improve sanitation and hygiene practices in a community. The approach tries to achieve behavior change in mainly rural people by a process of "triggering", leading to spontaneous and long-term abandonment of open defecation practices. It focuses on spontaneous and long-lasting behavior change of an entire community. The term "triggering" is central to the CLTS process: It refers to ways of igniting community interest in ending open defecation, usually by building simple toilets, such as pit latrines. CLTS involves actions leading to increased self-respect and pride in one's community. also involves shame and disgust about one's own open defecation behaviors. CLTS takes an approach to rural sanitation that works without hardware subsidies and that facilitates communities to recognize the problem of open defecation and take collective action to clean up and become "open defecation free".

Dry sanitation.

The term "dry sanitation" is not in widespread use and is not very well defined. It usually refers to a system that uses a type of dry toilet and no sewers to transport excreta. Often when people speak of "dry sanitation" they mean a sanitation system that uses urine-diverting dry toilet (UDDTs).

Ecological sanitation

Ecological sanitation, commonly abbreviated as ecosan (also spelled eco-san or EcoSan), is an approach to sanitation provision which aims to safely reuse excreta in agriculture.[33] It is an approach, rather than a technology or a device which is characterized by a desire to "close the

loop", mainly for the nutrients and organic matter between sanitation and agriculture in a safe manner. One of the aims is to minimise the use of non-renewable resources. When properly designed and operated, ecosan systems provide a hygienically safe system to convert human excreta into nutrients to be returned to the soil, and water to be returned to the soil, and water to be returned to the land. Ecosan is also called resource-oriented sanitation.

Emergency sanitation

Emergency sanitation is the management and technical processes required to provide sanitation in emergency situations. Emergency sanitation is required during humanitarian relief operations for refugees, people affected by natural disasters and internally displaced persons. There are three phases of emergency response: Immediate, short term and long term. In the immediate phase, the focus is on managing open defecation, and toilet technologies might include very basic latrines, pit latrines, bucket toilets, container-based toilets, and chemical toilets. The short term phase might also involve technologies such as urine-diverting dry toilets, septic tanks, and decentralized wastewater systems. Providing hand washing facilities and management of fecal sludge are also part of emergency sanitation.

Environmental sanitation

Environmental sanitation encompasses the control of environmental factors that are connected to disease transmission. Subsets of this category are solid waste management, water and wastewater treatment, industrial waste treatment and noise pollution control.

Fecal sludge management

Fecal sludge management (FSM) (or faecal sludge management in British English) is the storage, collection, transport, treatment and safe end use or disposal of fecal sludge. Together, the collection, transport, treatment and end use of fecal sludge constitute the "value chain" or "service chain" of fecal sludge management. Fecal sludge is defined very broadly as what accumulates in onsite sanitation systems (e.g. pit

latrines, septic tanks and container-based solutions) and specifically is not transported through a sewer. It is composed of human excreta, but also anything else that may go into an onsite containment technology, such as flushwater, cleansing materials (e.g. toilet paper and anal cleansing materials), menstrual hygiene products, grey water (i.e. bathing or kitchen water, including fats, oils and grease), and solid waste. Fecal sludge that is removed from septic tanks is called septage.

Improved and unimproved sanitation

Improved sanitation (related to but distinct from a "safely managed sanitation service") is a term used to categorize types of sanitation for monitoring purposes. It refers to the management of human feces at the household level. The term was coined by the Joint Monitoring Program (JMP) for Water Supply and Sanitation of UNICEF and WHO in 2002 to help monitor the progress towards Goal Number 7 of the Millennium Development Goals (MDGs). The opposite of "improved sanitation" has been termed "unimproved sanitation" in the JMP definitions. The same terms are used to monitor progress towards Sustainable Development Goal 6, they are a component of the definition for "safely managed sanitation service".

Lack of sanitation

Lack of sanitation refers to the absence of sanitation. In practical terms it usually means lack of toilets or lack of hygienic toilets that anybody would want to use voluntarily. The result of lack of sanitation is usually open defecation (and open urination but this is of less concern) with associated serious public health issues. It is estimated that 2.4 billion people still lacked improved sanitation facilities including 660 million people who lack access to safe drinking water as of 2015.

Onsite sanitation

Onsite sanitation (or on-site sanitation) is defined as "a sanitation system in which excreta and wastewater are collected and stored or treated on the plot where they are generated". The degree of treatment may be variable, from none to advance. Examples are pit latrines (no

treatment) and septic tanks (primary treatment of wastewater). On-site sanitation systems are often connected to fecal sludge management (FSM) systems where the fecal sludge that is generated onsite is treated at an offsite location. Wastewater (sewage) is only generated when piped water supply is available within the buildings or close to them. A related term is a decentralized wastewater system which refers in particular to the wastewater part of on-site sanitation. Similarly, an onsite sewage facility can treat the wastewater generated locally.

Sustainable sanitation

Sustainable sanitation is a sanitation system designed to meet certain criteria and to work well over the long-term. Sustainable sanitation systems consider the entire "sanitation value chain", from the experience of the user, excreta and wastewater collection methods, transportation or conveyance of waste, treatment, and reuse or disposal. The Sustainable Sanitation Alliance (SuSanA) includes five features (or criteria) in its definition of "sustainable sanitation": Systems need to be economically and socially acceptable, technically and institutionally appropriate and protect the environment and natural resources.

15.2. Importance of Hygiene

Hygiene, as defined by the WHO refers to “the conditions and practices that help maintain health and prevent the spread of diseases.”

This means more than just keeping ourselves clean. This means shunning all practices that lead to bad health. Throwing garbage on the road, defecating in the open, and many more. By adopting such a practice, we not only make ourselves healthier but also improve the quality of our lives.

Personal hygiene means keeping the body clean, consumption of clean drinking water, washing fruits and vegetables before eating, washing one’s hand, etc. Public hygiene refers to discarding waste and excreta properly, that means, waste segregation and recycling, regular disinfection and maintenance of the city’s water reservoir. Quality of hygiene in the kitchens is extremely important to prevent diseases.

Diseases spread through vectors. Say the vector is contaminated water as in the case of typhoid, cholera, and amoebiasis (food poisoning). By drinking clean water, we can completely eliminate the chances of getting diseases.

Some diseases are caused by pathogens carried by insects and animals. For e.g., plague is carried by rats, malaria, filarial, roundworms by flies and mosquitoes, etc.

Mosquitoes thrive in stagnant water and rats in garbage dumps and the food that is dumped out in the open. By spraying stagnant water bodies with kerosene or other chemicals, we can completely eliminate mosquitoes from our neighbourhood. If that is unfeasible, we can all use mosquito nets prevents us from mosquitoes while we're asleep. This poses a physical barrier for the mosquito.

Rats thrive on unsystematic waste disposal. By segregating the waste we can ensure that we don't leave food lying around for rats to eat. Close contact with sick people is also another way of contracting diseases.

A country has to strive to educate more doctors so that medical need of every citizen is taken care of. The importance of cleanliness should be inculcated in every citizen and this will in turn show in the cleanliness of the places we live in.

15.3. Importance of Sanitation

Sanitation is another very important aspect. Many of the common diseases mentioned earlier such as roundworms spread through the faeces of infected people. By ensuring that people do not defecate in the open, we can completely eliminate such diseases and even more severe ones such as the one caused by E. Coli.

The advancement in biology has given us answers to many questions, we are now able to identify the pathogen and treat an ailment accordingly.

Absence of basic sanitation facilities can:

1. Result in an unhealthy environment contaminated by human waste.
2. Contribute to the spread of many diseases/conditions that can cause widespread illness and death.

Sanitation infrastructure, poor hygienic practices, and unsafe drinking water negatively affect the health of millions of people in the developing world. Using sanitation interventions to interrupt disease pathways can significantly improve public health. Sanitation interventions primarily benefit public health by reducing the prevalence of enteric pathogenic illnesses. Health benefits are realized and accrue to the direct recipients of sanitation interventions and also to their neighbors and others in their communities. Billions of people have gained access to basic drinking water and sanitation services since 2000, but these services do not necessarily provide safe water and sanitation. Many homes, healthcare facilities and schools also still lack soap and water for handwashing. This puts the health of all people – but especially young children – at risk for diseases, such as diarrhoea. Safe water, sanitation and hygiene at home should not be a privilege of only those who are rich or live in urban centres,” says Dr Tedros Adhanom Ghebreyesus, WHO Director-General. “These are some of the most basic requirements for human health, and all countries have a responsibility to ensure that everyone can access them.

Health protection is that facet of public health that deals directly with some of the negative influences on people’s health and wellbeing, including radiation, infectious diseases, poisons, chemicals and environmental hazards. In many instances, it is up to the public health professionals to inform the public about what is bad for them, as well as how they can go about preventing themselves from becoming ill. Professionals might utilize television, radio and even billboards to get their messages across, including governmental organizations such as schools. By utilizing as many avenues as possible, these professionals ensure that their message gets across as quickly and as clearly as possible.

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