Course Name	**: Tropical / Infectious Diseases**
Course Code	**:** APBPH 3105
Course level	**: Level 5**
Course Credit	**: 4 CU**
Contact Hours	**: 60 Hrs**

Course Description
The Course gives a wide dissemination of knowledge about different kinds of diseases that public health practitioners are required to be informed about. These include Malaria, tuberculosis, syphilis, cholera, rabies, pneumonia, measles, anthrax, typhoid fever, hypertension etc. The course exacerbates the disease symptoms, signs, causes and effects to the human race.

Course Objectives
- To provide PHC students with a chance to understand the different interventions of the disease commonly known in public health.
- To further simplify the student's personal initiatives to prevent themselves from such diseases.
- To help students discover the misconceptions, myths and realities about the spread and transmission of these diseases.

Course Content
Introduction to disease in Public Health
Malaria
- Burden of Malaria
- Economical and socio-cultural impact of malaria
- Epidemiology of Malaria in Uganda or in your Country
- Intervention strategy for Uganda: the 2005-2010 National Malaria Control Plan
- Prevention of Malaria
- Emerging malarial issues

Tuberculosis and Multi Drug Resistant (T.B)
- Historical background of MDRTB
- Impact of tuberculosis on the community
- Causative agents of MDRTB
- Presentation and complications of MDRTB
- Treatment of MDRTB
- Drugs used for treatment of MDRTB

Syphilis
- Background of Syphilis
- Causes and prevention of the disease
- Primary syphilis
- Secondary syphilis

- Latent syphilis
- Prognosis and treatment of the disease
- Syphilis in HIV positive patients

Cholera

- Background of Cholera disease
- Causative agent
- Signs and symptoms
- Effects of cholera outbreak on community
- Treatment of cholera
- Cholera prevention and control

Rabies

Description of the disease
Stages of the disease
Diagnosis/treatment of rabies disease

Pneumonia

Description of Pneumonia
Causative agent
Signs and symptoms
How pneumonia is diagnosed
Prognosis and mortality
Treatment and prevention
Other public health preventive measures

Measles

- What is Measles
- How it spreads (transmissions)
- Signs of the disease
- People at risk
- Treatment
- Prevention of the disease

Anthrax

- Background of Anthrax
- What causes anthrax
- Signs and symptoms
- Diagnosis
- Prevention measures

Typhoid Fever

- What is Typhoid Fever
- Causes of typhoid
- Transmission and disease process
- Main forms of clinical presentation
- Management of typhoid fever
- Management of uncomplicated & typhoid fever

- Management of carriers
- Effects of typhoid on the government and community

Hypertension

- Definition of hypertension
- Epidemiology of hypertension
- Types of hypertension
- Risk factors for hypertension
- Causes of essential hypertension
- Causes of secondary hypertension
- Signs, symptoms and complications of hypertension
- Diagnosis of hypertension
- Control and treatment of hypertension
- Challenges in prevention, control and treatment
- Emerging issues regarding hypertension

Injuries

- Definition of injuries
- Types of injuries
- Grading of injuries
- Classification of injuries
- Impact of injuries
- What are known or acceptable methods of controlling injures
- Prevention of traffic road accidents and injuries
- The public health approach to injury prevention.

Mode of delivery Face to face lectures

Assessment
Course work 40%
Exams 60%
Total Mark 100%

DISEASES IN PUBLIC HEALTH

Introduction to diseases in Public Health
In this module, we will present the diseases with the highest public health importance; explain their causation, the complications, treatments and their public health implications. The chapter is divided into communicable and non communicable diseases, diseases related to lifestyle and injuries.

Summary

This course gives the learner overviews on the following aspects: background and burden of malaria in Uganda; social and economic impacts of malaria; causative agents and the mode of transmission; current interventions in the prevention and control of malaria in Uganda; recommended treatment regimens for adults, children and pregnant women; drug resistance in malaria (against the aminoquinolines) and finally, malaria interaction with HIV/AIDS.

Course work

The Ministry of Health of your country, together with WHO, are designing a new web page and need good information about non-communicable diseases to include on this page for public awareness and sensitising.

In not more than **1500 words,** give detailed and structured information (including illustrations to aid understanding and conveying the message in the best possible way to a broad public) on **ONE** of these diseases:

- **sickle cell disease**
- **breast cancer**
- **cervix cancer**
- **diabetes mellitus**

Gonorrhea

Communicable diseases

- Malaria Resource
- Tuberculosis and multi-drug-resistant-TB Resource
- HIV/AIDS Resource
- Syphilis Resource
- Whooping cough Resource
- Avian influenza (ex. of haemorrhagic fever) Resource
- Cholera Resource
- Rabies Resource

- Pneumonia Resource
- Measles Resource
- Chickenpox Resource
- Anthrax Resource
- Gonorrhea Resource
- Typhoid fever Resource
- Bilharzia (or schistosomias) Resource
-

Non communicable diseases

- Hypertension Resource
- Diphtheria Resource
- Coronary/Ischaemic heart disease Resource
- Injuries Resource
- Smoking Resource
- Sedentary living Resource
- Fatty diet Resource
- Alcohol and other drugs

Background

Background In almost all parts of Uganda, temperature and rainfall are sufficient to allow stable year round malaria transmission. Malaria is ranked the number one reported disease, causing high mobility and high economic and social impact. Malaria is highly epidemic in 95% of the country, an area that covers approximately 90% of the population of 29.4 million.

The remaining 5% consists of seasonal epidemic-prone malaria transmission in the highlands of the South-West and Mid-West, along the Eastern borders with Kenya and the North-Eastern border of Sudan. This area covers approximately 10% of the population.

Worldwide malaria causes approximately 500 million clinical cases and one million death each year (3,000 per day), 90% of them in Sub-Sahara-Africa. Because of the low levels of immunity to infection, children under the age of five are the group most heavily affected by malaria.

MALARIA

Burden of malaria in Uganda Some numbers:

- Malaria accounts for 20% of in-patient deaths.
- It has a case fatality rate of 3-5%.
- 23.4% of total discounted life years are lost.
- It accounts for 23% up to 11% of deaths among the under 5 years old in high and medium malaria transmission areas respectively.

- It is a major killer of refugees and internally displaced persons.

Economical and socio-cultural impact of malaria

Economic and socio-cultural impact of malaria First of all, there are the direct costs involved in the treatment seeking, the treatment and in some cases the funeral expenses. Prevention of the disease is expenditure. A poor malaria stricken family may spend up to 25% of its income on malaria treatment and prevention. Household surveys in Kabarole and Bundibugyo districts showed that the direct cost of treatment for an episode of suspected malaria averages 4,500 USh and 2000 USh in rural settings.

Moreover, by affecting families most during the rainy season when families can least afford to be sick, malaria interferes with their farm activities thus causing more poverty. It is estimated that malaria afflicted families average can only harvest 40% of crops harvested by healthy families.
In other economic fields, it generates a loss of household income through absenteeism from work. It is estimated that workers suffering from a malaria bout can be incapacitated for 5 to 20 days. A study in Apac, Kampala and Rukungiri districts showed that malaria was responsible for 54%, 33% and 50% respectively of absenteeism from work per month.

Malaria has also serious socio-cultural consequences. When it affects school-going children, it causes absenteeism from school thus affecting school performance. It is estimated that in endemic areas like Uganda, malaria may impair as much as 60% of school children learning ability.
Lastly, it is a huge burden on the health services. It is estimated that 40% of health facility expenditures in sub-Saharan African are spent on malaria.

- Epidemiology of malaria in Uganda

Epidemiology of malaria in UgandaParasite species All four common plasmodia species occur in Uganda, but Plasmodium falciparum is by far the most common contributing to 90-98% of the parasite population. The second most common species is Plasmodium malariae with 1-3% as a mono-infection, but it is more commonly found as a mixed infection with Plasmodium falciparum (up to 16% of childhood infections in highly endemic areas). Both Plasmodium vivax and Plasmodium Ovale are rare and do not exceed 1-1.5% of malaria cases.

Vectors.The most common vectors are Anopheles gambiae and Anopheles funestus with Anopheles gambiae being the dominant species in most places. Only during the dry seasons when permanent water bodies often are the most common breeding sites and in higher altitude areas is Anopheles funestus

found more frequently.

Intervention strategy for Uganda: the 2005-2010 National Malaria Control Plan

- Malaria prevention through the use of Insecticide Treated Nets (ITNs) with special emphasis on Long Lasting Insecticidal Nets (LLNs).
- Indoor Residual Spraying (IRS) with focus on low and epidemic prone areas
- Universal access to Artemisinin-based Combination Therapy (ACT) and improved diagnosis as well as severe malaria case management
- Emphasis on treatment and prevention of malaria in pregnancy including Intermittent Preventive Treatment of malaria during pregnancy (IPTp)
- Intensive Information, Education and Communication (IEC)
- Integration of malaria control into a balanced health system development with emphasis on human resource development
- Strong M & E

Prevention of malaria
The spread of malaria can be prevented in 4 ways;

1. The first prevention method is vector control. It involves reducing the mosquito population by killing mosquito larvae or adult through the use of insecticides or the destruction of breeding sites. Intensive vector control efforts in late 1940s successfully eradicated malaria in a wide geographic area including the USA, Europe and parts of Asia. However, such efforts were largely unsuccessful in Africa and most parts of Asia.
2. Secondly, you can reduce human mosquito contacts by the use of ITNs, protective clothing, repellents or indoor spraying of insecticides.
3. Thirdly, it is possible to prevent the establishment of infection through the use of Intermittent Preventive Treatment (IPT) or chemoprophylaxis. This use of IPT with prophylaxis anti-malarial drugs is the primary means of preventing malaria among pregnant women and children, who are particularly vulnerable to infection is. Traditionally chloroquine has been used for prophylaxis, but the wide spread emergence of chloroquine resistance dosing have made this approach ineffective. Currently, most IPT consists of 2 or 3 doses of sulfadoxine-pyrimethamine (SP). Ongoing studies are investing the efficacy of IPT regimens containing arteminisinin derivatives in combination with other antimalarial drugs. The recommended IPT regimen for pregnant women and children is: 500mg sulfadoxine + 25mg pyrimethamine (1 tab). 3 tablets are given at each dose. IPT-SP doses should not be given more frequently than once a month.
4. The fourth way of preventing malaria is to rapidly and effectively treat those who are infected in order to reduce the reservoir of infected people.

There is currently no vaccine for malaria although several trials are under way. But malaria is treatable and curable if detected early and if treatment is initiated promptly.

Emerging malarial issues

Emerging malarial issues

1. Wide spread drug resistance This drug resistance of the species has resulted from sub-optional dosing and overuse of drugs such as chloroquine. The chloroquine resistance is so widespread that it is no longer recommended for first-line treatment of uncomplicated falciparum malaria in most countries. Resistance to two other widely used drugs, SP and mefloquine is increasing as well. Because of the increasing resistance the WHO now recommends combination treatment based on artemisinin derivative for the first-line treatment of falciparum malaria in Africa and Asia.
The current recommended antimalarial co-formulation include:
Artemether + lumefantrine
Mefloquine + artesunate
Amodiaquine + artesunate
Sulfadoxine-pyrimethamine SP+ artesunate

2. Malaria and HIV interaction
The interaction between malaria and HIV infection is complex and subtle. There are several potential ways in which malaria and HIV infection could interact, co-infection could affect the progression or clinical manifestation of either conditions, infection with either malarial parasite or HIV could facilitate transmission of the other, co-infection could affect treatment outcomes, and there may be toxicities or interactions between the drugs used to treat the different conditions.

Effects of HIV on malaria
By compromising the acquired immunity of adults or children in endemic areas, HIV infection increases the incidence of malaria and the clinical severity of infection. Studies throughout Sub-Saharan Africa have found out that co-infection with HIV approximately doubles the risks of parasitemia and clinical malaria.
HIV infected patients with severe immuno-suppression experience more severe malaria than their non-infected counterparts and they require more frequent treatment for uncomplicated malaria. The negative interaction between the two is most apparent in pregnant women.

Effect of HIV on malaria treatment
Co-infection with HIV reduces the efficacy of malaria treatment. A study in Kenya found out that co-infected patients with CD4 cells counts of < 200 cells

/ÂµL who were treated with SP had significantly lower rates of parasite clearance in 28 days following treatment.
HIV infection also reduces the efficiency of arteminsinin-based malaria treatment. A retrospective study in Uganda found that adults infected with HIV responded worse to malaria treatment that their uninfected counter-parts in the 28 days following treatment.
HIV infection in absence of antiretroviral treatment may interfere with the effectiveness of standard IPT regimens such as SP.

Anti-malarial and antiretroviral drug interactions
At the moment there are no documented clinical or pharmacological interactions between anti-malarial drugs and antiretroviral drugs. However, there are several theoretical interactions of which clinicians should be aware. Protease inhibitors and non-nucleoside reverse transcriptase inhibitors (NNRTIS) are the primary classes of antiretroviral drugs that have the pharmacokinetics potential to interact with anti-malarial drugs.

3. Weak health systems
Even with a good policy in place, health system weaknesses, particularly human resource shortages and commodity procurement and supply, can slow policy implementation and malaria diagnosis, especially in high transmission settings that already present a challenge. The inadequate or unreliable supply of medication is a major challenge that threatens the health system overall. Also weak referral systems continue to impede care.

TUBERCULOSIS AND MULTI DRUG RESISTANT TB

While tuberculosis is a deadly infection caused by the mycobacteria tuberculosis, multi-drug-resistant tuberculosis (MDR-TB) can be defined as tuberculosis which, as a result of strains emerged through spontaneous mutation of the bacilli, is resistant to isoniazid and streptomycin, the two most powerful existent anti-TB drugs.
The cause of this lies in an ineffective treatment of the original TB, due to patients missing doses or not completing the treatment, or doctors prescribing inappropriate handling of the case.

- Historical background of MDRTB
- Impact of tuberculosis on the community
- Causative agents of MDRTB
- Presentation and complications of MDRTB
- Prevention and control of MDRTB
- Treatment of MDRTB
- Drugs used for treatment of MDRTB

Historical background

Before the discovery of specific antibiotics for the treatment of tuberculosis there was no cure. Mortality of those with pulmonary disease was about 50%

By the end of the 1930s surgeons were providing some means of treatment for tuberculosis by the various surgical procedures attempting to obliterate the cavities which formed in the lungs of seriously ill tuberculosis patients by the lung itself

By the 1950s the introduction of drug therapy was considerably reducing the number of patients and there was 98% chance of the cure. The difficulties of ensuring this, especially in resource poor countries, have resulted in an increasing incidence of the tubercle bacteria resistant to the most effective drugs. This is called multi-drugresistance tuberculosis.

Impact of tuberculosis on the community

Impact on the patient
Patients with tuberculosis are stigmatized because they are isolated by the community. They cease to be productive, as generally they are too weak and too isolated to work. The isolation and stigmatisation leads often to the breaking up of the family.

Impact on the health sector
Drug resistant tuberculosis increases the cost and the duration of treatment. The new rapid method of detecting drug resistant tuberculosis is helpful, but they are too costly and not seen as a priority in market oriented economics of the pharmaceutical industry. They leave many families very poor after having raised a lot of money to save the life of a family member.

In conclusion drug resistant tuberculosis can be prevented if patients play their part of taking drugs as they should and well trained health workers who know how to administer tuberculosis drugs and are authorized to do so, are the only ones to administer these drugs.

Causative agents

Tuberculosis is caused by a bacteria bacillus called mycobacterium tuberculosis and occasionally by mycobacterium bovis and mycobacterium Africanum. These organisms are also known as tubercle bacilli because they cause lesions called tubercles.

Other causes of resistant tuberculosis:

- previous treatment for tuberculosis especially if prolonged

- contact with another patient known to have drug resistant tuberculosis
- immigration from one area with a high incidence of drug resistance
- HIV seropositivity
- Substance abuse like tobacco
- Homelessness, sleeping in many places
- Incorrect administration of the drug
- Poor drug quality
- Failure of the patient to take the drug consistently

Presentation of multi drug resistant tuberculosis

- Chronic cough is almost always present. The cough stays and does not go away even after treatment.
- Sputum, usually made of pus
- Haemoptysis, sometimes it can be large other times it is small.
- Pain in the chest, usually of the pleuritic type, although this is not common
- Shortness of breath. Sometimes this comes early in the disease. If the disease is severe there is a large pleural effusion, otherwise it only comes after years when most of the lungs are destroyed.
- Fever and sweating, especially at night
- Loss of weight
- Sometimes mild fever
- sometimes anaemia
- Ordinary pneumonia

Complications

- Disease of the organ. The infection by the myco-tuberculosis organisms leads to destruction of the organs and formation of pus
- Pneumonia
- Kidneys may be affected causing urinary tract infection or blood in the urine
- When the disease reaches the vertebrae there will be paralysis or loss of sensation in the legs and patient may not be able to to pass urine because of pressure on the spinal cord
- Chronic arthritis in cases of tuberculosis arthritis
- Tuberculosis osteomyelitis
- Heart infection, generally the pericardium

Prevention and control

Prevention is better than cure. In order to ensure that the patient is taking medication correctly the use of directly observed therapy (DOT) is indicated, where the patient is seen swallowing his or her medication under the eyes of trained supervisor.

To prevent this drug resistance emerging, the medical worker should ensure that immunotherapy is avoided. Another measure is the isolation of the effected patients until they are out of the infectious stage. In case of bad side effects like itching, rash, blister and nausea, the patient should be told to go back to the health centre instead of just giving up, thus causing resistance of the drug.

Only well trained health workers who have good knowledge on tuberculosis and have been authorized should be allowed to administer these drugs because any mistake done by a health worker in administering these drugs can cause resistance.

As said before, proper counselling should be done to patients and family members so that the patient takes drugs the way they should be taken. Furthermore, health workers should sensitize everyone who comes at the health centre if symptoms develop and even give BCG to other children and adults who have a negative tuberculin test.
The health workers also should do all they can to stop HIV infection spreading because this affection of the immune system is the main reason why tuberculosis is becoming more and more common today.

Treatment

- *Do not* start treatment unless sputum is positive for Acid-fast Bacillus (AFB)
- The lymph gland aspirate or biopsy should be positive for tuberculosis
- Also treatment should not be started unless the patient has suspected severe tuberculosis and would die before test results could be obtained or transfer was possible.
- Also treatment should be not be started unless tuberculosis has been diagnosed by a medical officer in another way.

Management of the patient

- Arrange for a place where it is convenient for the patient to get the directly observed treatment (DOTS) by health workers, unless he or she is to be admitted to the health centre to start treatment immediately.
- Start patient and family education about the disease, the treatment, and the possible side effects of the treatment and when to return if worried about possible side effects. Explain that he will be cured if he takes his treatment and will probably die and infect his family and friends if drugs are not taken.
- Arrange for check sputum examination for acid-fast bacillus after 2 months of treatment, after 5 month of treatment and at the end.
- Check the patient's attendances for treatment every month
- Arrange for home visit if the attendance for treatment is not good

- Give a leaflet about tuberculosis and its treatment to those who can read and are interested to know more about their condition.
- Arrange treatment as near as possible to the patient's home or his work. If needed, time his clinics so that the patient does not have to miss work and try not to keep the patient waiting.
- In primary stages treating the patient should be isolated. Carefully tell the patient and give him a card, the date and place of his next attendance. If there is a local calender different from the standard international calender give him the date in the local calender so that he will understand better.
- Check on his/her personal problems for example job problems, marriage, what his neighbours will say, give him or her kind and friendly advice about any problem and if possible get a friendly personality nurse to do the counselling.
- If he is not having direct observed treatment when he comes back for a new supply of drugs, remember to check the number of pills left over. This will tell you whether he has taken all the doses, ask him in a sympathetic way why he has not. This will help you give the right advice.
- If the patient does not get better or does not return for review, the best and quickest way to get the patient is by home visit to persuade him to return. That is why a patient has to leave his contacts and home address.

Treatment is divided into three parts

1. Short term intensive chemotherapy daily for 8 weeks (2months) with four (or at least three) drugs to try to kill most of the Mycobacterium tuberculosis organisms during this time.
2. Maintenance treatment for 4 or 6 or 10 or more months (depending on what drugs are available for short term intensive chemotherapy and maintenance therapy) to kill the rest to the organisms especially the resistors.
3. Surgical treatment, drug therapy for resistant tuberculosis carried a succeed rate considerably lower than for sensitive disease. 60%-70% cure compared with 95% surgery is sometimes useful adjunct. If a disease is confined to one or the most two lobes, lobectomy offers a better chance of than continued drug treatment.

Drugs used for treatment

First line drugs

Essentials

- Isoniazid-kills the bulk of the bacteria
- Rifampicin-Eliminates the resistant bacteria

Other drugs

1. Pyrazinamide
2. Ethambutol
3. Streptomycin

New drugs

- Rifamycins
- Rifabutin
- Rifapentine

Second line drugs

Old drugs

- Ethinonamide
- cycloserine
- capremycin
- amikacyn
- kanamycin
- PAS
- Thiocetazone

New drugs

- Quinolones
- ofloxacin
- ciprofloxacin
- sparfloxacin
- Macrolides
- clarithromycin
- clofazimine
- amoxicillin
- clavulanic acid

Because of the emergence of more drug resistance cases world wide the current recommendation is to give two drugs from first line drugs (others) pyrazinamide and ethamboutol, in addition to isonaizid and rifampcine until culture and sensitivity results are available. Patients should not be started on two drugs alone.

In patients who have had previous treatment, that is to say they are not new to the disease; a more complex regimen may be needed initially. It is important that the four drug regimens are continued until culture results are available.

In the immigrants streptomycin resistance is so common that this should not be included in the regimen even if the patient has not been exposed to it previously.

If the patient has had previous treatment with isoniazid, refampicin, ethambutol and pyrazinamide he should be given an injectable drug as a mickacin, at least one old drug such as athionamide and one of the new drugs as a ciprofloxacin. Cycloserine could be used as a fourth drug if required. Thus the potentially drug-resistant patient will be started on six drugs. The danger of adding a single drug to a regimen already being given will therefore be avoided.

HIV/AIDS refer to your HIV Mgt module
SYPHILLIS
Background

There is some discussion between historians if syphilis existed in Europe before the travels of Columbus to America. Probably there was a form of tertiary syphilis present before the bacteria that cause non-venereal syphilis and jaws arrived from America in the 1490s, causing a major syphilis epidemic in Naples, Italy in 1494.

The discovery of penicillin had a great impact on syphilis throughout the world in the late 1940s. This led to the decline in the number of cases seen in sexually transmitted infection clinics. Nonetheless, since 1998 infectious syphilis has increased substantially in towns like London, Manchester and Brighton mostly as a result of homosexual transmission. The incidence increased from 122 to 1193 cases between 1996 and 2002, as cited in The ABC of sexually transmitted infections of Michael Adler.

Syphilis presents a major clinical problem and WHO estimates that 12 million new cases of infectious syphilis are diagnosed worldwide each year and most of these cases are in South and South-East Asia and Sub-Saharan Africa.

- Cause and presentation of syphilis
- Prognosis and treatment of the disease
- Syphilis in HIV positive patients
- References

Cause and presentation of the disease
Syphilis is caused by a spirochete bacterium called Triponemapallidum. The manifestation of syphilis varies according to its stages: primary, secondary and latent syphilis.

Primary syphilis
Lesions are at the site of inoculation. These are usually painless and may be genital or extra genital. There are enlarged inguinal lymph nodes.

Secondary syphilis
Appears 4 to 8 weeks after appearance of the primary lesion. Generalised lesions affecting both the skin and mucous membranes. They are usually symmetrical.
May be associated with malaise, fever, anorexia and glomerulonephritis (a disease of the kidneys).
Bacteraemia (increased bacteria circulation in blood), leading to hepatitis (inflammation of liver), meningitis, iritis (inflammation of the iris of the eye) and papilloedema (a swelling of the blood vessels of the retina at the back of the eye).
Generalised lymphadenopathy (enlarged lymph nodes).
Alopecia (loss of body hair).

Latent syphilis
Even without treatment, some of the patients (about 65%) have syphilis in the latent stage and present no symptoms or signs of syphilis. Latent syphilis is divided into early and late stages. The distinction is made after two years of being infected without showing the symptoms and is important for the treatment.
Serological tests will be positive, but normal radiological tests will give no results.

10% of the cases may develop neurosyphilis (nervous system involvement) and 10% cardiovascular syphilis.
Prognosis and treatment of the disease
This depends on the stage of the disease and the degree of tissue damage in cardiovascular and neurosyphilis. Adequate treatment of all the stages halts the progression of the disease.
Treatment
Benzathine 2.4 mega units as a single dose in mild cases and 3 doses in one week interval for severe cases.
Procaine penicillin 600, 000 IU and 900,000 IU.
Doxycycline 100mg orally twice a day for 14 days.

Nowadays modern antibiotics can be used in treating syphilis.
Syphilis in HIV positive patients
Syphilis enhances the acquisition and transmission of HIV. The signs and symptoms of syphilis can be mistaken for the clinical features of HIV infections. The signs shared by syphilis and HIV includes the following:

- Generalised lymphadenopathy
- Skin rashes
- Alopecia
- Oral ulceration
- Cognitive impairment

- Meningitis
- Cranial nerve palsies
- Myelopathies (spinal cord disorders)
- Uveitis

WHOOPING COUGH

Whooping Cough is a highly contagious bacterial infection caused by Bordetella Pertussis. It is one of the most serious childhood diseases. The disease is transmitted by direct contact or droplet spread and has fastidious growth requirements. The incubation period of pertussis is usually about 7 days, with a range of 5 to 21 days. A non-immune person is susceptible to pertussis at any age, but characteristically it is a disease of children under 7 years old. There is probably no trans-placental immunity, and 40% of all deaths from pertussis occur among infants less than 5 months of age. The period of infectivity begins 7 days after exposure and extends for 3 weeks after the onset of the paroxysm [outburst, spasm,convulsion, fit] of coughing. After 6 weeks patients may be considered non infectious.

The occasional second attacks do not represent true pertussis; they are usually caused by B.parapertussis or B.bronchiseptica, neither of which shares cross-immunity with B.pertussis. They are milder illnesses.

- Clinical manifestations
- Complications and effects on the community
- Treatment and prevention
- References

Clinical manifestations
The clinical course of B. pertussis is usually divided into three stages: catarrhal, paroxysmal and convalescent[recuperative or restorative, recovery].

The *catarrhal stage* lasts about 1-2 weeks. It begins as a typical upper respiratory tract infection, with low grade fever, coryza, sneezing, lacrymation, irritability and a dry, poorly productive cough. The cough worsens and after a week begins to occur paroxysmal.
The *paroxysmal stage* lasts from 4 to 6 weeks. There are explosive bursts of coughing in rapid succession during which the child cannot breathe. The coughing is followed by a sudden, long inspiration that rushes air into the emptied lung and produces the crowing, high-pitched whoop. One paroxysm of coughing may follow another until the child is able to cough up a thick, tenacious mucous plug. During the coughing spell the child appears cyanotic or livid, the tongue protrudes, and the eyes bulge. After the attack the child vomits, perspires profusely and appears lethargic and exhausted. Peri-orbital oedema, conjunctivalhaemorrhages and epistaxis may also be present. In infants under 6 months of age, the characteristic whoop may not be present.

The uncomplicated *convalescent stage* is marked by cessation of whooping and vomiting and an improvement in appetite and mood. The paroxysms are milder and occur less frequently, but coughing may persist for several weeks. If a secondary respiratory infection has developed, recurrent paroxysms of coughing and whoops may reappear repeatedly for many months.

Complications

The most frequent complication is pneumonia, usually caused by secondary bacterial invaders. Atelectasis and pneumonia predispose to the late development of bronchiectasis. Otitis media is commonly seen among infants. A convulsion is a serious complication of whooping cough; predisposing conditions are brain damage due to hypoxia or focal haemorrhage and alkalosis produced by repeated vomiting.

Effects on the Community

Whooping cough is an infectious disease and can increase the mortality rate if it is not picked up and treated. It can also lead to disabilities like brain damage as mentioned above. It can also lead to injuries when the child is having a coughing fit. As a secondary effect, it leads to the parents of the child being distressed since their child is unwell.

Treatment

The treatment of B. pertussis requires both specific and general measures. Erythromycin is effective in modifying the course of uncomplicated whooping cough. Late in the course of the disease it may still decrease the number of bacteria, possibly reducing communicability. The use of sero-therapy in the form of hyper immune gamma globulin is controversial. Apnea and encephalopathy are probably toxin related complications of pertussis. These, as well as atelectasis and pneumonia, necessitate the availability of expert medical and nursing care for the sick patient with the disease.

Prevention

The value of active immunization against B.pertussis is well established. Although untreated patients may be contagious for approximately 4 weeks, antimicrobial therapy (erythromycin) reduces this period, even if coughing persists. Treatment of exposed family contacts with erythromycin estolate is recommended.

AVIAN INFLUENZA (ex. of haemorrhagic fever)

Background

Avian influenza also called ‘bird flu’ is a highly contagious viral infection, caused by a virus belonging to the family Orthomyxoviridae. It’s caused by type A strains of influenza viruses that normally infect only birds, and sometimes pigs.

Avian influenza has two forms; one that causes mild illness in birds, and another, known as "highly pathogenic avian influenza (HPAI)" that is extremely contagious and rapidly fatal for infected birds. The HPAI strain involved in current outbreaks is called H5N1. It was first recognized in 1997 in Hong Kong. At that time millions of chickens were slaughtered after the virus was found to cause disease in people exposed to infected birds. 18 people got infected of which six died. Fortunately, the virus was not able to spread from person to person, and the outbreak was halted in Hong Kong by slaughter of the chickens.

In January 2004, a major new outbreak of H5N1 avian influenza surfaced again in Vietnam and Thailand's poultry industry, and within weeks spread to ten countries and regions in Asia, including Indonesia, South Korea, Japan and China. Intensive efforts were undertaken to slaughter chickens, ducks and geese (over 40 million chickens alone were slaughtered in high-infection areas), and the outbreak was contained by March, but the total human death toll in Vietnam and Thailand was 23 people.

In Africa, the first outbreak of H5N1 highly pathogenic avian influenza (HPAI) was confirmed at Kaduna, Nigeria, on 8 February 2006. Within three months, seven other countries on the continent, Burkina Faso, Cameroon, Côte d'lvoire, Djibouti, Egypt, Niger and Sudan, were infected. More recently Ghana and Togo became infected. The origin of the introduction of the disease to Nigeria and the other infected countries is still unknown, owing to lack of adequate tracing of the movements of poultry and poultry products and lack of reliable epidemiological data from the affected countries.

In Uganda, there has not been any outbreak of Avian Influenza reported however, preventive measures have been put in place to contain the virus (H5N1). Two state of the art laboratories, one in Entebbe and the other in the faculty of Veterinary Medicine at Makerere University have been put in place to analyse any samples from the various water bodies for any contamination.

The influenza viruses occur naturally among birds. Wild birds carry the viruses in their intestines, but usually do not get sick from them.

Infected birds shed influenza virus in their saliva, nasal secretions, and faeces. Domesticated birds may become infected with avian influenza virus through direct contact with infected waterfowl or other infected poultry, or through contact with surfaces e.g. cages or materials e.g. feeds, that have been contaminated with the virus.

Infection with avian influenza viruses in domestic poultry causes two main forms of disease that are distinguished by low and high extremes of virulence. The low pathogenic form may go undetected and usually causes only mild symptoms (e.g. ruffled [disheveled, messy, tangled, windswept feathers] while

the highly pathogenic form spreads more rapidly through flocks of poultry. This form may cause disease that affects multiple internal organs and has a mortality rate that can reach 90-100% often within 48hours.

Types and subtypes
There are three types of influenza viruses: A, B, and C. Only influenza A viruses are further classified by subtype on the basis of the two main surface glycoproteins hemagglutinin (HA) and neuraminidase (NA). Type A includes three sub types (H1N1, H2N2 and H3N2) that have been associated with widespread epidemics and pandemics; type B has been associated with regional or widespread epidemics and type C has been associated with sporadic cases and minor localized outbreaks. The virus type is determined by the antigenic properties of the two relatively stable structural proteins, the nucleoprotein and the matrix protein. Influenza A subtypes and B viruses are further classified by strains. Of the 15 subtypes known, only subtypes H5 and H7 are known to be capable of crossing the species barrier from birds to humans.

Transmission
Human influenza is transmitted by inhalation of infectious droplets and droplet nuclei, by direct contact, and perhaps, by indirect (fomite) contact, with self-inoculation onto the upper respiratory tract or conjunctival mucosa. The relative efficiency of the different routes of transmission has not been defined. For human influenza A (H5N1) infections, evidence is consistent with bird-to-human, possibly environment-to-human, and limited, no sustained human-to-human transmission to date.

Bird to human
By plucking and preparing of diseased birds; handling fighting cocks; playing with poultry, particularly asymptomatic infected ducks; and consumption of duck's blood or possibly undercooked poultry are sure modes of transmission.

The virus can be shed in the droppings of migratory birds since they are natural carriers and is able to survive for three months in cool temperatures. The virus can survive in water at 0Â°C for more than 30 days and at 22Â°C for up to 4 days. If a person working closely with these animals inhales dust particles containing the virus or by other means, they could develop the bird flu

In countries where live birds (e.g. chickens, geese, turkeys) are sold in markets along with pigs or raised near pigs, the possibility of the virus recombining with other subtypes is greater. This is because both human and avian viruses can infect pigs. If a pig is infected with both viruses at the same time, different parts of the avian and human viruses can mix with each other. Later, the avian virus that has picked up some genes from the human form of the influenza virus is able to more easily cause the conditions in humans.

Human to human

Human-to-human transmission of influenza A (H5N1) has been suggested like child-to-mother transmission, intimate contact without the use of precautions also mentioned, but so far no case of human-to-human transmission by small-particle aerosols has been identified. Recently, intensified surveillance of contacts of patients by reverse transcriptase polymerase-chain-reaction (RT-PCR) assay has led to the detection of mild cases, more infections in older adults, in northern Vietnam. Findings suggest that the local virus strains may be adapting to humans.

Environment to human

Given the survival of influenza A (H5N1) in the environment, several other modes of transmission are theoretically possible. Oral ingestion of contaminated water during swimming and direct intranasal or conjunctivae inoculation during exposure to water are other potential modes, as is contamination of hands from infected fomites and subsequent self-inoculation. The widespread use of untreated poultry feaces as fertilizer is another possible risk factor.

Incubation

The incubation period of avian influenza A (H5N1) may be longer than for other known human influenzas. In most cases it occurs within two to four days after exposure; recent reports indicate similar intervals but with ranges of up to eight days.

Disease progression

The Avian Influenza A virus is the H5N1 virus responsible for the Avian Influenza disease. Highly pathogenic avian influenza A (H5N1) virus is able to cross the species barrier and cause infection and illness in humans. The pathogenesis of the disease is not completely understood. The main clinical feature of the disease is, still, severe pneumonia often complicated by Acute Respiratory Distress Syndrome (ARDS). Although mild cases and sub-clinical illness have been reported, most patients experience severe illness, and overall the case fatality rate among laboratory confirmed cases remains as high as sixty percent.

Symptoms of Asian influenza

In birds

The most common symptoms of avian flu in birds are: ruffled feathers, reduced

egg production, respiratory distress. In some cases, domestic birds may die the same day symptoms appear.

In humans
Most patients have initial symptoms of high fever (typically a temperature of more than 38 °C) and an influenza-like illness with lower respiratory tract symptoms. Upper respiratory tract symptoms are present only sometimes. Unlike patients with infections caused by avian influenza A (H7) viruses, patients with avian influenza A (H5N1) rarely have conjunctivitis. Diarrhoea, vomiting, abdominal pain, pleuritic pain, and bleeding from the nose and gums have also been reported early in the course of illness in some patients. Watery diarrhoea without blood or inflammatory changes appears to be more common and may precede respiratory manifestations by up to one week

The most common symptoms of avian influenza in humans are: fever, cough, sore throat, muscle aches, eye infections and pneumonia.

Diagnosis

Early stages of influenza, when transmission first begins, lack distinguishing clinical symptoms and thus require a biochemical test. Current detection technologies are PCR (polymerase chain reaction), viral culture, Immunoassays however these are a little slow (minimum time 2 hours)
Laboratory diagnosis
Samples
Identification of the agent
Live birds tracheal swabs and cloacae swabs or faeces
Dead birds organs and faeces
Serology
Clotted blood samples or
Serum

Procedures
Identification of the Agent
Inoculation of 9-11-day-old embryonated chicken eggs followed by:
1.Haemagglutination immunodiffusion test to confirm the presence of influenza A virus
2.Subtype determination with monospecific antisera
3.Strain virulence evaluation: evaluation of the intravenous pathogenicity index (IVPI) in 4-8-week-old chickens

Serology. Tests available:
ELISA
Detects antibodies to all AI virus, does not distinguish subtypes
Only suitable for testing chicken and turkey serum
Within 1 week of infection, antibodies are detected in more than half the

specimens.

AGID(Agar Gel Immunodiffusion test)

As for ELISA does not distinguish AI subtypes

Within one week of infection, antibodies are detected in more than half the specimens.

HI (Haemagglutination Inhibition test)

Serotype specific test

Test available for each H subtype

HI titres are positive a few days later than ELISA or AGID, titres persist long after infection

Standard test for all avian species

IFT (Immunofluoresence test)

1.Able to detect antibodies to specific N-subtype

2.Can be used to detect infection in vaccinated birds if a heterologous vaccine is used.

RT-PCR (Reverse-transcriptase polymerase chain reaction)

Able to detect influenza virus at very low levels

The presence of subtype H5 or H7 can be confirmed by using H5 or H7 specific primers.

Treatment

Early recognition of patients and timely administration of an influenza-specific antiviral agent using standard protocols are essential for further evaluation of the effectiveness of antivirals. Amantadine or rimantadine started within 48 hours of onset of Influenza A illness and given for approximately 3 and 5 days reduces symptoms and virus in the respiratory secretions.

During treatment with either drug, drug-resistant viruses may emerge late in the course of the treatment and may be transmitted to others. Cohorting patients on antiviral therapy should be considered. Patients should be watched for development of bacterial complications and antibiotics administered.

Effects on community

Farmers and other people working with poultry, as well as travellers visiting affected countries, have a higher risk for getting the bird flu. Handling an infected bird can cause infection. People who eat raw or undercooked poultry meat are also at an increased risk for avian influenza. Highly infective avian flu viruses, such as H5N1, have been shown to survive in the environment for long periods of time, and infection may be spread simply by touching contaminated surfaces. Birds who recover from the flu can continue to shed the virus in their faeces and saliva for as long as 10 days.

Preventive measures

The preventive measures adopted in countries free from H5N1 HPAI include:
Selective or total bans on the importation of poultry and poultry products from infected countries.

Quarantine, stamping-out and active surveillance, while poultry vaccination was carried out in Co´ted'lvoire and Egypt.
Culling (killing) will prevent the spread of the avian flu to other birds (and farms), and also minimize the risk of human infection.

Public health preventive measures:
Keep poultry in closed poultry houses to prevent contamination of wide areas. This has been problematic in the Avian flu outbreaks in South-east Asia where many affected farms allowed their poultry to range freely.
Keep wild birds and their faeces away from poultry and poultry feed. Wild birds, particularly migratory waterfowl, have been implicated as carriers or reservoirs of the virus. They are more resistant to the disease, thus they can go harbouring and shedding the virus for long periods of time. Many of the avian flu outbreaks suggest that the point source of infection originated from wild birds passing on the virus to domestic birds.
Seal poultry house attics and cover ventilation openings with screens.
Thoroughly and routinely clean all equipment, vehicles, including service vehicles, clothing and footwear before and after coming into contact with poultry. Birds shed large amounts of virus through their faeces and nasal passage (nasal spray, saliva).
Ensure proper hygiene practices for all persons coming into contact with poultry.
Maintain high sanitation standards in and around poultry houses
Isolate or avoid introducing new birds into existing poultry flocks if their health status is unknown.
Limit access to poultry houses, including farm workers, feed suppliers, poultry veterinarians, catching crews, sawdust and shavings suppliers, agricultural service personnel and casual visitors.
Avoid using water in poultry houses contaminated with faeces from wild birds.
Ensure thorough cleaning and disinfection for all cages transporting birds.
Maintain a log of all visitors coming into contact with poultry.

CHOLERA

Background of cholera disease

Populations all over the world are reported to have at one time or the other sporadically been affected by devastating outbreaks of cholera. Records from Hippocrates (460-377 BC) and Galen (129-216 AD) already described an illness that might well have been cholera, and numerous recorded hints indicate that a cholera-like malady[PROBLEM] was also known in the plains of the Ganges River since antiquity[ANTIQUE,ANCIENT TIMES,RELIC]. Other recorded evidence of cholera refers to cholera in 1563 in a medical report from Indian subcontinent where the disease has probably existed for thousands of years. Modern knowledge about cholera, however, dates only from the beginning of the 19th century when researchers began to make progress towards a better

understanding of the causes of the disease and its appropriate treatment. The first pandemic, or global epidemic, started in 1817 from its endemic area in South-East Asia and subsequently spread to other parts of the world. The 1st and subsequent pandemics inflicted a heavy toll, spreading all over the world before receding.

In 1961, the 7th cholera pandemic wave began in Indonesia and spread rapidly to other countries in Asia, Europe, Africa, and finally in 1991 to Latin America, which had been free of cholera for more than one century. The disease spread rapidly in Latin America, causing nearly 400 000 reported cases and over 4000 deaths in 16 countries of the Americas that year.

Cholera has often occurred in outbreaks or epidemics; seven pandemics (worldwide epidemics) of cholera have been recorded between 1817 and 2003. The first of these pandemics was recorded in 1817, and the world is currently experiencing the seventh cholera pandemic, which is reported to started in Indonesia in 1961 and spread rapidly in Asia, Europe, and Africa, and reached South America in 1991. Still the seventh pandemic has not receded; on the contrary, cholera has now become endemic in many parts of the world with particular impact in the developing countries..

However, poor surveillance and fear of international stigmatisation and sanctions have always led to under reporting of official numbers by affected countries. For example, in 2001, 58 countries officially notified World Health Organisation (WHO) of a total of 184 311 cases and 2728 deaths but, due to considerable under-reporting, the true global figures are estimated to have been closer to 1 million cases. Estimates of global cholera-specific mortality are believed to be 100 000 to 130 000 deaths per year, with most of the deaths occurring in Asia and Africa. Case fatality rates (CFRs) vary greatly from country to country depending probably on how efficient the existing health infrastructures are able to cope with the epidemic.

Since 2005, the re-emergence of cholera has been noted in parallel with the ever-increasing size of vulnerable populations living in unsanitary conditions. The number of cholera cases reported to WHO during 2006 rose dramatically, reaching the level of the late 1990s. A total of 236 896 cases were notified from 52 countries, including 6311 deaths, an overall increase of 79% compared with the number of cases reported in 2005. This increased number of cases is the result of several major outbreaks that occurred in countries where cases have not been reported for several years. It is estimated that only a small proportion of cases - less than 10% - are reported to WHO. The true burden of disease is therefore grossly underestimated. These reported increasing spread of cholera in recent years may reflect a lack of effective international quarantine enforcement by some countries which also have inefficient public water supply systems and inadequate sanitary regulations, the international mobility of

carriers in the world's population, and the quick transport of contaminated food and water by ships and aircraft.

Causative agent

Cholera is an infection caused by the bacteria Vibrio cholera and the principal site affected is the gastrointestinal tract. Cholera is rarely transmitted directly from one person to another. It is the contamination of food or water with faeces of an infected person that is the main source of the disease. Thus, people become infected usually by drinking water or eating food contaminated by the bacteria. Poor sanitation as well as poor personal and domestic hygiene practices have been noted as other sources of contamination and infection. Cholera outbreaks are closely linked to inadequate environmental management. Rapid urbanisation without adequate sanitation and access to clean drinking water has also contributed a lot to the persistence of the epidemic. Thus typical at-risk areas include peri-urban slums, where basic infrastructure is not available. Some agricultural husbandry practices such use of human faeces contaminated fertilizers in fruits and vegetables gardens have also been associated with the disease especially where such plants are eaten raw or insufficiently cooked. Cholera bacteria also live in warm, saline water and can infect persons who eat raw or undercooked seafood obtained from such waters. Although cholera vibrios may persist for only a short time in grossly polluted aquatic environment, faecal contamination from victims of epidemics and the carriers may continue to reinforce their population in water. Anyone is susceptible to cholera infection, but infants, children, and the elderly are more likely to die from the disease because they become dehydrated faster than adults. Though there is no particular season in which cholera is more likely to occur, wet or rainy seasons are of particular concern especially in slum areas of developing countries due to the ease with which contaminated water effluent may flow and meander to wider areas than during dry seasons.

Other very important risk factors for cholera epidemic outbreaks include overcrowded living conditions, unstable political and environmental conditions such as wars, famines and floods that lead to displaced populations and the breakdown of service delivery (such as health and water) infrastructure. However, it is important to stress that the belief that cholera epidemics are caused by dead bodies after disasters, whether natural or man-made, is false. Nonetheless[nevertheless, however, on the other hand, even so], such conditions that cause disruption of water and sanitation systems or massive displacement of population to inadequate and overcrowded camps can increase

the risk of transmission, should the pathogen be present or introduced. Hence, the focus of epidemics/pandemics has shifted to developing countries over the last century, where such conditions have become more of a norm.

Healthy carriers of V. Cholera, who though usually rare, may also pose a health risk to others as another potential source of infection. These symptomless carriers excrete vibrios intermittently with the duration of pathogen discharge being relatively short, averaging 6 to 15 days with a maximum period between 30 to 40 days. Chronic recuperative or convalescent carriers have been observed to shed vibrios intermittently for periods of 4 to 15 months. Survival of vibrios in the aquatic environment relates sharply to various chemical, biological and physical characteristics of a given water source. The viability of V. cholera in surface waters has been observed to vary from 1h to 13 days. Although cholera vibrios may persist for only a short time in grossly polluted aquatic environment, faecal contamination from victims of epidemics and the carriers may continue to reinforce their population in water.

Signs and symptoms

There is usually a 1 to 5 day incubation period, from the ingestion of the bacterium to the appearance of the first signs of the infection, and the disease runs its course in 2 to 7 days depending on the strain of the Vibrio ingested, and the immunity status of the person affected. Most people exposed to cholera don't become ill and never know they've been infected. Yet because they shed the bacteria in their stool for seven to 14 days, they still have the potential to infect others. The great majority of people who become sick experience mild or moderate diarrhoea that's often hard to distinguish from diarrhoea caused by other problems especially those with strong body immunities.

Steps in the pathogenicity of cholera include colonization of the small intestinal mucosa and elaboration of the cholera enterotoxin, which causes hyper-secretion of fluids and electrolytes through stimulation of the cell, thereby leading to the body to lose both water and electrolytes leading to dehydration.

Only about one in 10 infected people develop the typical signs and symptoms of cholera, which include:

Severe, watery diarrhoea. Diarrhoea comes on suddenly. It's often voluminous, composed of mucus and dead cells, and has a pale, milky appearance that resembles water in which rice has been rinsed (rice water stool). What makes cholera diarrhoea so deadly is the loss of large amounts of fluids in a short time.

Nausea and vomiting; Occurring in both the early and later stages of the disease, vomiting may persist for hours at a time.

Muscle cramps. These result from the rapid loss of salts such as sodium, chloride and potassium.

Dehydration. This can develop just hours after the onset of symptoms â€” far more quickly than in other diarrhoeal diseases. Depending on how much body

weight has been lost, dehydration can range from mild to severe; a loss of 10 percent or more of total body weight indicates severe dehydration. Signs and symptoms include irritability, sluggishness or lethargy, sunken eyes, a dry mouth, extreme thirst, dry and shrivelled skin that's slow to bounce back when pinched into a fold, little or no urine output, low blood pressure, and an irregular heartbeat (a condition known as arrhythmia).
Shock. Hypovolemic shock is one of the most serious complications of dehydration. It occurs when low blood volume causes a drop in blood pressure and a corresponding reduction in the amount of oxygen reaching the tissues of an affected patient. If untreated, this severe hypovolemic shock can cause death in a matter of minutes.

In general, children with cholera present the same signs and symptoms as adults do, but they may also experience extreme drowsiness or even coma, fever, and convulsions. Death may occur within 24 hours of onset of signs and symptoms unless prompt medical treatment is given to the patient.

Prognosis

According to WHO, among people developing symptoms, 80% of episodes are of mild or moderate severity. Among the remaining cases, 10%-20% develop severe watery diarrhoea with signs of dehydration. If untreated, as many as one in two people may die. Patients with milder cases of cholera usually recover on their own in three to six days without additional complications. They may eliminate the bacteria in their faeces for up to two weeks. With prompt fluid and electrolyte replacement (rehydration) which is simple and inexpensive, the death rate in patients with severe cholera is less than 1%. Untreated, the death rate can be greater than 50%.

However, though this situation may present itself in developed countries, in developing countries where infrastructure for medical service delivery is poor, the outcome may be quite different; and usually an outbreak is followed by a state of panic and apprehension. Thus the difficulty in treating severe cholera does not lie in not knowing how to treat it but rather in getting medical care to the sick in underdeveloped and sometimes inaccessible areas of the world where medical resources are limited. Cholera however is a very treatable disease if timely and correct diagnosis is made.

Effect of cholera outbreak on community

Cholera remains a global threat to public health and one of the key indicators of social development. Cholera outbreaks can have far reaching consequences on the community directly or indirectly by disrupting social and economic structure, and impeding development in affected communities, especially in developing countries where the disruption is usually greater:
Cholera can cost governments billions of money to eradicate. This would mean that resources have to be diverted from other sectors for which they were budgeted to counter the outbreak.
Absenteeism by the workforce caused by cholera adversely affects industrial

and economic output. Peoples income will thus be negatively affected.
Cholera outbreaks can adversely affect tourism and thus affect tax revenues (productivity losses for business and individuals due to the illness will decrease tax revenues).
Cholera outbreaks may lead to loss of trade. Outbreaks set into motion unjustified panic-induced reactions by other countries that include curtailing or restricting travel from countries where a cholera outbreak is occurring, or imposition of import restrictions on certain foods. For example, a cholera outbreak in Peru in 1991 cost the country US$ 770 million due to food trade embargoes and adverse effects on tourism.
Cholera outbreaks are usually followed by restrictions on movement (quarantine), isolation of the sick, and stigmatisation of the sick.
The worst effect on community especially in developing counties where families are closely associated is the loss of a loved one especially the breadwinner. Burying of cholera victims may not allow cultural rituals as it has to be done by medical personnel, and this may leave the communities indisposed.

Treatment of Cholera

The first and crucial measure in the treatment of cholera is a correct and rapid diagnosis of cholera. This can be made by examining a fresh stool sample under the microscope for the presence of V. cholera bacteria. Cholera can also be diagnosed by culturing a stool sample in the laboratory to isolate the cholera-causing bacteria. In addition, a blood test may reveal the presence of antibodies against the cholera bacteria. In areas where cholera occurs often, however, patients are usually treated for diarrhoea and vomiting symptoms as if they had cholera without laboratory confirmation.

The key to treating cholera lies in preventing dehydration by replacing the fluids and electrolytes lost through diarrhoea and vomiting. No matter which method is used, immediate treatment is critical because death from cholera can occur within hours. Without rehydration, approximately half of people with cholera die; with treatment, the number of fatalities drops to less than one percent. The discovery that rehydration can be accomplished orally revolutionized the treatment of cholera and other, similar diseases by making this simple, cost-effective treatment widely available throughout the world.

The World Health Organisation (WHO) thus has established guidelines for treating cholera that can be used in the most severe cases and circumstances. WHO in conjunction with UNICEF has thus developed an inexpensive oral replacement fluid containing appropriate amounts of water, sugar, and salts that is used worldwide. The solution, called WHO/UNICEF ORS, is available as a powder that can easily be reconstituted in boiled or bottled water. Early rehydration can save the lives of nearly all cholera patients. The majority of patients - up to 80% - can be treated adequately through the administration of oral rehydration salts (using the WHO/UNICEF ORS standard sachet). In cases of severe dehydration, replacement fluids must be given intravenously. Patients should be encouraged to drink plenty of fluids when they can keep liquids

down and eat when their appetite returns. Recovery generally takes three to six days.

Rehydration usually occurs in two stages. The initial phase treats existing dehydration; in the maintenance phase, fluids are continually replenished until diarrhoea stops. The amount of solution needed to maintain hydration varies greatly, depending on the severity of dehydration and the degree of diarrhoea. But most people require large amounts of fluids, especially at the start of treatment. People who have trouble drinking ORS, either because of frequent vomiting or the sheer volume of fluids may need infusion through their veins (intravenous treatment) using preferably Ringer lactate.

In addition to rehydration, people who are very sick may benefit from antibiotics, which can cut the length of the illness in half. Adults may be given the antibiotic tetracycline to shorten the duration of the illness and reduce fluid loss. The World Health Organization however recommends this antibiotic treatment only in cases of severe dehydration. If antibiotics are overused, the cholera bacteria organism may become resistant to the drug, making the antibiotic ineffective in treating even severe cases of cholera. Tetracycline is not to be given to children whose permanent teeth have not come in because it can cause the teeth to become permanently discoloured. Other antibiotics such as ciprofloxacin and erythromycin may be given to speed up the clearance of V. cholera from the body.

Oral rehydration therapy however remains the most important means of treatment for cholera patients.

CHOLERA PREVENTION AND CONTROL

Measures for the prevention and control of cholera have not changed much in recent decades, and mostly consist of:

Provision of clean and safe water ; The need for safe drinking water is a need that binds all of humanity into a single, global community.

Safe disposal of human excreta; This is of the utmost importance in control of infectious and other communicable disease including cholera. Because of the importance of the safe disposal of human excreta, the building of appropriate sanitation systems often is considered synonymous with improving sanitation and controlling cholera.

Effective primary health care education programme; This should include personal hygiene, food preparation and health education (especially in developing countries where resources may be inadequate, particularly in rural communities). In particular, systematic hand washing should be taught. Once an outbreak is detected, the usual intervention strategy is to reduce mortality by ensuring prompt access to treatment and controlling the spread of the disease. Experience has shown that unless there is an effective primary health care education programme that addresses issues such as domestic activities

related to the storage and use of water, and sewage and excreta disposal, and personal hygienic practices, the installation of improved sanitation facilities alone may not result in improved health. The mere material improvement of water supplies would doubtless prove to be less effective than if people were advised by means of health education of the sources of their disease problems and how to avoid them. Primary health care education is thus a vital component in prevention of cholera.

Surveillance and prompt reporting; these contributes to the rapid containment of cholera epidemics. In many endemic countries, cholera is a seasonal disease, occurring every year usually during the rainy season. Surveillance systems can provide an early alert to outbreaks, which should lead to a coordinated response, and assist in the preparation of preparedness plans. As part of an integrated surveillance system, an efficient cholera surveillance system can also improve the risk assessment for potential cholera outbreaks. Comprehensive surveillance and data generation are of paramount importance to guide the preventive rather than reactive planning of interventions so as to adapt them to each specific situation. Understanding the seasonality and location of outbreaks will provide guidance for improving cholera control activities for the most vulnerable. This will also contribute to developing indicators for appropriate use of oral cholera vaccines.

Adaptation of recommended control methods; WHO has developed and published such recommended measures, including standardised case management, has proven effective in reducing the case-fatality rate.

Adoption of a multi-sectoral coordination approach; since cholera prevention and control is not an issue to be dealt by the health sector alone, a comprehensive multidisciplinary approach should be adopted for dealing with a potential cholera outbreak. Water, sanitation, education and communication are among the other sectors usually involved. A better understanding of the socio-economic, environmental and public health consequences of water supply and sanitation related diseases obtainable through better monitoring surveillance systems may help the public and policy makers understand the value of microbiologically safe water as well as improved sanitation facilities.
Harness political commitment and community involvement; Policy makers and leaders must make appropriate policies that ease such control measures above to be implemented. They must also allocate a substantial amount of resources to emergency preparedness and infrastructure development. The community leaders must be actively involved in appropriate health and proper hygiene education.

Burying rather than cremating those that have died of cholera;Because cholera is one of the few infectious diseases that can be spread by human remains (through faecal matter leaking from corpses into the water supply), emergency workers who handle human remains are at increased risk of

infection. It is considered preferable to bury corpses rather than to cremate them, however, and to allow survivors time to conduct appropriate burial ceremonies or rituals. The remains should be disinfected prior to burial, and buried at least 90 feet (30 m) away from sources of drinking water.

Use of cholera vaccines; Two types of vaccines do exist, i.e. parenteral (administered by injection) and enteral (administered orally). However, the use of the parenteral whole cell cholera vaccine has never been recommended by WHO due to its low protective efficacy (of about 60 days) and the high occurrence of severe adverse reactions or reactogenicity, though it has been used in some countries.

Currently available oral cholera vaccines (OCV) are safe and offer good protection (over 70%) for an acceptable period of time (at least one year). The OCV is usually suitable for travellers especially when travelling to highly cholera prone areas. This vaccine has proven safe and effective (85-"90% after six months in all age groups, declining to 62% at one year among adults) and is available for individuals aged two years and above. It is administered in two doses 10-15 days apart and given in 150 ml of safe water. Its public health use in mass vaccination campaigns is relatively recent. Within the past few years several immunization campaigns were for example carried out with WHO support just like other public health intervention tools; and in 2006, WHO published official recommendations for OCV use in complex emergencies. WHO recommends OCV use for populations to limit the risk of occurrence of cholera outbreaks in displaced populations in endemic areas, and spread and incidence of cholera during an outbreak?

Though OCV have been recommended by WHO as a public health intervention measure in emergencies, and many vaccines are under development, it is important to note that vaccines are cholera strain specific, and therefore a given vaccine has a limited scope of application. Correct diagnosis of the target V. cholera strain before administration of a particular vaccine is thus important.

RABIES

Rabies is a zoonotic viral disease, fatal to the central Nervous System; and its caused by a Neurotropic virus consisting of non segmented, negative stranded Ribonucleic acid (RNA) contained within a bullet shaped envelope. It belongs to genus Lyssa virus and the family Rhabdoviridae. Rabies common sites of entry into humans are through the skin, or mucus membranes where the virus is delivered into the muscle and subcutaneous tissue through biting, licking or scratching by a rabies virus infected animal.

It presents into two forms thus the classic form or encephalitic (furious form) in the central nervous system causing a cute encephalomyelitis. Here the symptoms include: pharyngeal spasms, hydrophobia, and hyperactivity leading to paralysis, coma and death. Rabies is distinguishable by neurotropism, neuroinvasiveness and impaired

functions, malaise, fever headache.

Paralytic rabies is characterised by development of prominent and flaccid muscle weakness, excitability, convulsions, delirium. Death results from both neural dysfunctions due to dramatically inhibited synthesis of proteins required for maintaining neuronal functions.

Clinical stages of rabies disease progression

These depend on the extent of the bites, amount of secretion, and proximity to the central nervous system. Disease transmitted through bites close to the brain progress more rapidly than disease transmitted through bites on the lower extremities.

Stages of the disease

a) Its incubation stages ranges from 10 days to 1 year.
b) Prodrome stage, this occurs 2 to days after the exposure and can last for 1 to 2 weeks and itâ€™s characterized by non specificflu like symptoms such as fever, malaise, headache and nausea.
c) Acute neurologic syndrome occurs 7 or 10 days after the onset of prodrome and includes dysarthia, dysphasia, excessive salivation, vertigo, agitation, visual and auditory hallucinations, hydrophobia secondary to painful contractions of pharyngeal muscles and polyneuritis.
d) Coma occurs 7 or 10 days after the onset of acute neurologic syndrome and its characterized by hydrophobia, prolonged apnea, generalized flaccid paralysis, seizures and coma with acute respiratory collapse.
e) Death may follow 2 or 3 days after the onset of paralysis.

Epidemiology of rabies disease
The primary route of rabies virus transmission to humans is through animal bite; hence the animal is a reservoir for rabies virus and the opportunity for human animal interaction.

Canid species are the main vector in the transmission of rabies to humans' e.g. Rabid bats especially silver- haired bats.

Other rare routes may include; handling of infected carcasses, consumption of raw infected meat and inhalation of aerosolized rabies in caves inhabited by millions of bats.
Few cases of human to human transmission of rabies have resulted from the transportation of infected corneas.

Diagnosis / Treatment of rabies disease

In humans and before death, observe the virus specific fluorescent material in skin biopsy specimens, isolating the virus from the patients saliva, or detecting the presence of anti-rabies antibodies in the serum or cerebral spinal fluid (CSF) of patients who have not been immunised.

The basic principles behind rabies prophylaxis are the removal of free virus from the body both by washing and neutralisation.

Performing induction of a rabies virus specific immune response in the exposed individual before rabies virus can replicate in the central nervous system by administering of both passive and active vaccination.

In passive vaccination, Rabies immune Globins (RIG) from adults who have been immunized with rabies vaccine is administered to previously unimmunized people so as to passively impart antibodies.

Active rabies vaccines currently administered include; nerve tissue derived vaccines (NTV), high quality cell culture vaccines produced under stringent quality control and lower â€“ quality cell culture vaccines that do not adhere to FDA regulations.

Nerve tissue derived vaccines (NTV) are produced from brain tissue of animals infected with a fixed strain of rabies virus. Brain tissue is harvested; the virus is inactivated and then diluted to a concentration of 25 % of brain tissue.

Nerve tissue derived vaccines are extremely painful and can cause severe neurologic adverse reactions due to presence of myelinated tissue in the vaccine; unfortunately nerve tissue vaccines are the most widely used prophylaxis for rabies.

Optimal rabies vaccine today is human diploid cell vaccine (HDCV) which is a type of cell culture vaccine produced in human fibroblasts. Treatment is generally unsuccessful when administered after the patient becomes sick.

PNWUMONIA

What is pneumonia?

Pneumonia is an inflammatory illness of the lungs. Frequently it is described as lung parenchyma/alveolar inflammation and abnormal alveolar filling with fluid. Pneumonia is an infection of one or both lungs.

Prior to the discovery of antibiotics, one third of all people who developed

pneumonia subsequently died from infection. In the United States pneumonia is the sixth leading cause of death.

Causative agent

Pneumonia is usually caused by bacteria, viruses, or fungi. The most common cause of pneumonia is the bacterium â€œStreptococcuspneumoniaeâ€□ or â€œpneumococcusbacteriumâ€□. Another bacterium is Klebsiellapneumoniae and Hemophilusinfluenzae bacteria that cause pneumonia in people suffering from chronic obstructive pulmonary, disease or alcoholism.

The other causative agent of pneumonia is the virus as already stated. Viral infections can be caused by adenoviruses, rhinovirus, influenza virus (flu), respiratory syncytial virus, and para influenza virus. Fungal infections that can lead to pneumonia include histoplamosis, coceidiomycosis, blastomyxosis, aspergillosis and cryptococeosis

Pneumonia can also result from chemical or physical injury to the lungs. its cause may also be officially described as idiopathic -that is, unknownâ€“ when infectious causes have been excluded.
How do people â€œcatchpneumoniaâ€□
Breathing in small droplets that contain the organism that can cause pneumonia contracts some cases of pneumonia. These droplets get into the air when a person infected with these germs coughs or sneezes.

In other cases, pneumonia is caused when mouth, throat, or nose secretions inadvertently enter the lung. During sleep, it is quite common for people to aspirate secretions, from the mouth, throat, or nose. Normally the bodyâ€™s reflex response (coughing back up the secretion) and immune system will prevent the aspirated organisms from causing pneumonia. However, if a person is in a weakened condition from another illness, a severe pneumonia can develop.

Once organisms enter the lungs, they usually settle in the air sacks of the lung where they rapidly grow in number. This area of the lung then becomes filled with fluid and pus as the body attempts to fight off the infection
Signs and symptoms

In most people who develop pneumonia there is:
Symptom of a cold

Followed by a high fever (sometimes as high as 104degrees F.)
Shaking chills
A cough with sputum production (usually discolored or phlegm and sometimes bloody).
May become short of breath.
Chest pain may develop if outer pleural aspects of the lung are involved. This pain is usually sharp and worsens when taking a deep breath, known as pleuritic pain.

In other cases, there can be a slow onset of symptoms. A worsening cough, headaches, and muscle aches may be the only symptoms. At times, the individuals skin color may change and become dusky or purplish (a condition known as â€œcyanosisâ€) due to their blood being poorly oxygenated.

Other possible symptoms are loss of appetite,fatigue,blueness of skin, nausea, vomiting,mood swings and joint pains or muscle aches. Less common forms of pneumonia can cause other symptoms; for instance, pneumonia caused by legionella may cause abdominal pain and diarrhoea while pneumonia caused by tuberculosis or pneumocystis may cause weight loss and night sweats.

Children and babies who develop pneumonia often do not have any specific signs of a chest infection but develop a fever, appear quite ill, and can become lethargic. Certain groups of people are considered to be at particularly high risk for the development of pneumonia and the U.S center for disease control and prevention recommended vaccination for the following groups:
People aged 65 or older and those over two years of age who have problems with their lungs, heart, liver, kidneys.
People age 65 or older and those over two years of age with health problems like diabetes, sickle cell diseases, alcoholism or HIV/AIDS.
Persons over two years of age who are taking any treatments that weaken the bodys immune system.

How pneumonia is diagnosed?

Pneumonia may be suspected when the doctor examines the patient and hears coarse breathing or crackling sounds when listening to a portion of the chest with a stethoscope. There may be wheezing, or the sounds of breathing may be faint in a particular area of the chest. A chest x-ray is usually ordered to confirm the diagnosis of pneumonia.

Sputum samples can be collected and examined under the microscope. If bacteria or fungi that cause pneumonia are present, they can often be detected by this examination.

A blood test that measures white blood cell count (WBC) may be performed either. An individuals WBC count can often give a limit as to the severity of the pneumonia and whether it is caused by bacteria or a virus. An increased

number of neutrophils (one type of WBC), is seen in bacterial infection, whereas an increase in lymphocytes, is seen in vi+ral infections.

Bronchoscopy; thin, flexible lighted viewing tube is invested into the nose mouth.

Prognosis and mortality

With treatment, most types of bacterial pneumonia can be cleared within two to four weeks. Viral pneumonia may last longer and in cases where the pneumonia progresses to blood poisoning (bacteremia), just over 20% of sufferers will die. The death rate or mortality also depends on the underlying cause of the pneumonia and also in regions of the world without advanced health care systems, pneumonia is even deadlier. Limited access to clinics and hospitals, limited access to x-rays, limited antibiotic choices, and inability to treat underlying conditions inevitably leads to higher rates of death from pneumonia.

Treatment and prevention

For streptococcus pneumoniae, antibiotics are often used in the treatment. They include penicillin, amoxicillin and clavulanic acid also macrolide antibiotics including erythromycin, azithromycin, (zmax) and clarithromycin (Biaxin).

Two vaccines, pneumococcal conjugate vaccine (pcv7) and the pneumococcal poly-saccharide vaccine (pp23) pneumovax are available to prevent pneumococcal disease. Pcv7 is usually used in immunisation of infants while pneumorax is recommended for adults at increased risk for developing the diseases.

Klebsiellapneumonial and hemophilusinfluenzae bacteria that cause pneumonia in people suffering from chronic obstructive pulmonary, disease or alcoholism use of the second and third generation antibiotics like cephalosporins amoxicillin, and clavulanic acid, fluoroquinolones and sulfamethozaxole.

Viral infections can be caused by adenoviruses rhinovirus, influenza virus (flu), respiratory syncytial virus, and para influenza virus. These pneumorial usually resolve overtime with the bodyâ€™s immune system fighting off the infection.

Fungal infections that can lead to pneumonia include histoplamosis, coceidiomycosis, blastomyxosis, aspergillosis and cryptococeosis. Each fingus has specific antibiotic treatment, among which are amphoterianB, fluconazde (diflucan), penicillin and sulfonamides.

Since it is airborne, in many hospitals, patients with this infection are placed in contact isolation. Their visitors are often asked to wear gloves, masks and

gowns. It is therefore very important to wash your hands thoroughly and frequently to limit **further spread.**

Other public health preventative measures

Appropriately treating underlying illnesses (such as AIDS) can decrease a personsrisk of acquiring pneumonia.
Smoking cessation is important not only because it helps to limit lung damage, but also because cigarette smoke interferes with many of the body's natural defences against pneumonia.
Testing pregnant women for group B Streptococcus and chlamydia trachomantis, and then giving antibiotic treatment if needed reduces pneumonia in infants.
Vaccination as earlier mentioned, is important in preventing pneumonia in both children and adults.

MEASLES

What is measles?

It is a contagious disease caused by the paramyxo virus of the genus morbilli. According to the World Health Organization (WHO) measles is an acute illness. It grows in the cells that line in the lungs and the cells that line the back of the throat. It's a human disease not known to occur in animals.

"German measles" is an unrelated condition caused by the Rubella virus.

- Transmission, signs and risk group
- Treatment and prevention

How it spreads (transmissions)

Measles is spread through respiration (contact with fluid from an infected persons nose and mouth either directly or through aerosol transmission) and is highly contagious 90% of people without immunity sharing the same house with an infected person can catch the disease.

The incubation period is usually 4-12 days (during which that are no symptoms). The infected persons or people remain contagious from the appearance of the first symptoms until 3-5 days after the rash appears.

Signs (symptoms)
The first sign is usually high fever that begins approximately 10 to 12 days after exposure and lasts 1-7 days. The patient develops a runny nose, cough, red and watery eyes and small white spots inside the cheeks. After seven days, the rush develops usually on the face and upper neck and then after three

days, the rush spreads all over the body.

People at risk
Un-immunised young children are at highest risk for measles and it is complications include death, deafness or blindness. However any person who has not been immunized with vaccine or through experiencing the disease can become infected. It is a big killer children disease.

Treatment
It has no specific drug for treatment;
All children diagnosed with measles should receive two doses of Vitamin A supplements given 24 hours apart
Antibiotics for treatment of eye and ear infections and phenomenon
Nutritional fluid support and treatment of dehydration with oral rehydration solution.
Maintain bed rest and provide quite activities for the child. If there is sensitivity to light, keep room daily lit.
Remove eye secretions with saline or water and encourage the child not to rub the eyes
Administer anti pruritic medication and
A cool mist vaporizer can be used to relief cough
Isolate the patient until the fifth day

Prevention
Generally two doses of live measles vaccine are recommended
Immunisation at nine months is recommended one time for all persons born and who lack immunity to measles.
Using measles mumps rubella (MMR) vaccine is necessary.

CHICKEN POX

Definition and aetiology
Chickenpox, also known as varicella, is a highly contagious acute viral disease that causes an itchy rash that appears in crops. It is caused by Varicella-zoster virus (VZV) which is a herpes virus. Over 90% of non immune individuals will develop chickenpox following exposure

Herpes virus infections were known as early as ancient Greek times. Hippocrates described the cutaneous spreading of herpes simplex lesions and scholars of Greek civilization coined the word herpes in reference the creeping or crawling nature of the herpetic skin lesions. In Shakespeare's famous play 'Romeo and Juliet', there ia an allusion to recurrent herpes simplex lesions and transmission of the disease. However it was not until 1893 when Vidal recognized that human transmission of Herpes Simplex infection from one individual to another was possible.

Historical accounts were not clear on the difference between small pox lesions and those of VZV and it was not until the late eighteenth century that Heberden proposed a clinical method to differentiate the two diseases. Later in 1888, Von Bokay intimated that chickenpox and herpes zoster were due to the same causal agent an idea which was confirmed by Weller and Stoddard.

- Epidemiology, clinical presentation and prognosis
- Effects on community, treatment and preventionS

Epidemiology
The disease which has a world wide distribution affects mostly children between 5-10 years of age. Outbreaks are common in schools and emergency settings. In the tropics a higher proportion of those suffering from the disease are adults. The source of infection is an infected person and transmission is through direct skin-to-skin contact and droplet nuclei produced during maneuvers like coughing and sneezing. Nosocomial transmission is also well recognized. Mother to child transmission is possible in utero.

Clinical presentation
The disease begins with a prodromal phase occurring approximately 2 weeks from exposure and lasting for 1-2 days. This phase is characterised by fever (up to 38.8 degrees Celsius), abdominal pain, sore throat, and malaise. Within 24 hours of the prodromal phase, a characteristic itchy rash develops starting on the trunk and spreading towards the head, arms and legs in the next 10 days. The rash may also spread to other sites like the mouth and genitalia. Each lesion begins as a red papule and progresses to a blister, then pustule and finally to a scab. The lesions, which may be up to 300 in number, are at different stages of development. In congenital varicella syndrome, the new born presents with dermatomal scars, microcephaly, muscle and bone defects, visual problems and mental retardation.

Prognosis
The disease is usually self-limiting and lifelong immunity generally follows the primary disease. In some cases the immune system does not totally clear the virus from the body (the virus is shielded by hiding in the skin sensory nerve cell bodies) and subsequent compromise in immunity leads to release of the virus from the reservoirs causing herpes zoster (shingles) with a life time risk of 10-20%. In congenital disease, babies born to mothers who are infected shortly before delivery may develop potentially life threatening infections. Complications may occur secondary to the primary disease. They include: secondary bacterial infections caused by either Staphylococcus or Streptococcus bacteria; pneumonia especially in adults; neurological complications and Reyeâ€™s syndrome which is associated with associated with the administration of aspirin to children. Other complications include:

hepatitis, kidney disease, ulcers of the intestinal tract, and inflammation of the testes (orchitis). Severe viral pneumonia may occur in adults. Congenital disease may occur if the mother develops chicken pox during pregnancy

Effects on the community

The disease has a number of community effects ranging from stigma to restriction on movements. Congenital disease may lead to mental retardation hence affecting academic and social function. Loss of loved ones and bread winners affects the social, psychological and economic function of the community

Treatment

Treatment involves the administration of acyclovir within 24 hours of the rash, antihistamines like diphenhydramine, loratadine or cetirizine to relieve itching and paracetamol or ibuprofen for fever. Secondary bacterial infection should be treated using appropriate antibiotics. Neonatal infection may be treated with varicella zoster immune globulin.

Prevention

Prevention is through: active vaccination with the chickenpox vaccine at 12 to 18 months old and a booster shot at 4 to 6 years. Vaccine efficacy is 70% to 85% at preventing mild infection and over 95% at preventing moderate to severe forms of the disease; administration of VZ immunoglobulin within 96 hours of exposure to an infected person. Avoiding contact with active cases is also important.

ANTHRAX

Background

What is Anthrax

Anthrax is a life threatening infectious disease that normally affects animals especially ruminants such as goats, cattle, sheep, horses. Other animals like cats, dogs, swine and rats are resistant to the infection. Anthrax is common in areas where people raise livestock and where public health programs are lax. Animals get anthrax by grazing on soils contaminated with anthrax spores. It can be transmitted to humans by contact with infected animals and their products or by biological warfare. Anthrax does not spread from person to person.

What causes anthrax?

Anthrax is caused by bacterium called Bacillus anthraces. This germ was discovered in 1850 by a German physician known as Robert Koch. Unlike most bacteria, these exist in a dormant form as spores in the soil, animal carcasses, feces and animal products. However, under the microscope, these germs look like large rods. These spores have a long life span, as long as 48years and are very difficult to destroy. The spores them selves have no significant damage to tissue, they can however lead to disease by entering broken skin, being inhaled

and being eaten. Once in the body the spores germinate to form disease-causing bacteria.

Signs and Symptoms
In the body, anthrax bacteria produce a powerful toxin which is responsible for the illness. Signs and symptoms vary depending on how a person was infected. Anthrax can infect humans in 3 ways:

1.Through broken skin. This is called cutenous anthrax. This is the most common form of infection. It starts as a red brown raised itchy swelling on the skin that looks like an insect bite. With in 1-2days it develops into a boil-like sore and then a painless ulcer with a hard and dark part at the centre called a malignant pustule. The infection can also cause swelling of the lymph glands near the site. There may be headache, fever, nusea and vomiting. Death is rare with proper treatment as 80% of the treated patients survive. The 20% die because infection spreads to other parts of the body. The illness usually resolves in about 6 weeks.

2.By ingestion. This is called gastro intestinal anthrax. It is as a result of eating undercooked, contaminated meat. The bacteria infect the wall of the intestinal tract causing severe inflammation. The infection then spreads throughout the body via the bloodstream (septicemia). The first signs are nausea, loss of appetite, vomiting and fever. These are later followed by abdominal pain, vomiting blood and severe diarrhea. Gastrointestinal anthrax results in death in 25%-60% cases.

3.By inhalation of the spores. This is called inhalation anthrax. It is the most dangerous and the greatest bioterrorism threat. It is an occupational hazard for people who sort wool (wool sorters disease). Spores are inhaled, transported through the air passages into the tinny sacs (alveoli) in the lungs. The spores are then picked up by scavenger cells (macrophages) in the lungs and are transported through small vessels (lymphatic) to the glands or lymph nodes in the central chest cavity (Mediastinum). Here, they germinate into active, reproducing bacteria. The bacteria produce toxins which cause severe bleeding and tissue death (necrosis). From there, the toxin spreads to the adjacent lungs and to the rest of the body by way of the blood stream. Damage to the central chest cavity and lungs can cause chest pain and difficulty in breathing. The toxins are the primary causes of destruction of tissue, haemorrhage and death. The first symptoms are difficult to perceive but are more like flue or common cold. In a few days the illness worsens, there may be severe respiratory distress, shock, coma and eventually death. This type of anthrax usually results in death in1-2 days after the start of severe symptoms.

Symptoms usually appear with in 7 days after exposure to the infection.

Diagnosis

The history including the occupation of the person infected is important. Anthrax is diagnosed by cultures and smears from the infected tissues. Material is collected from presumed sites of infection i.e.:

-Skin sores for cutaneous anthrax
-Sputum from patients with inhalation anthrax
-Stool samples for gastrointestinal anthrax
-Chest X rays may show characteristic changes in and between lungs.
-If the organism has spread to the nervous system, spinal fluid may demonstrate the organism. If they have spread throughout the body they can be demonstrated in a blood sample.
-Nasal swabs can be used to determine if some one has been exposed to anthrax by inhalation but can not confirm infection.

Treatment

In most cases, early treatment can cure anthrax. Anthrax of the skin can be treated with common antibiotics such as penicillin, tetracycline, erythromycin and ciprofloxin. Once it has spread to all parts of the body, it calls for a medical emergency. It can however be treated with a combination of penicillin and streptomycin as an early intervention.

Prevention

When travelling to areas where anthrax is common and vaccination levels of animal herds is low, avoid contact with livestock and animal products. Avoid eating meat that has not been properly slaughtered and cooked. Do not buy items made of animal fur or wool.
A vaccine is available for people at high risk for work-related exposures such as the veterinarians, laboratory technicians, and employees of textile mills processing wool or fur.
Put in place measures for preventing bioterrorism attacks and prepare to deal with it if it occurs.

Glossary

Alveoli are tinny air sacks in the lungs where the exchange of oxygen and carbon dioxide takes place.
Bio-terrorism/biological war fare- terrorism using biological agents
Lymph nodes/glands are small rounded/bean shaped masses of the lymphatic tissue, surrounded by a capsule of connective tissue. They filter the lymphatic fluids and store special cells that can trap cancer cells or bacteria that could be travelling through the body in the lymphatic fluid.
Lymphatic tissue - A part of the body's immune system that helps protect it from bacteria and other foreign entities.
Lymphatics are small thin channels similar to blood vessels that do not carry blood but collect and carry tissue fluid called lymph from the body to

ultimately drain back into the blood stream.
Malignant- tending to be severe and being able to invade and destroy nearby tissue.
Pus is a mixture of inflammatory cells and liquid
Pustule is a small collection of pus in the upper layer of the skin (epidermis) or beneath it in the dermis. It frequently forms in sweat glands or hair follicles.
Sputum- the mucus and other matter brought up from the lungs, bronchi and trachea that one may cough up and spit out or swallow.
Tissue refers to any group of cells that perform specific functions.

TYPHOID FEVER

Typhoid fever is caused by Salmonella typhi, a Gram-negative bacterium. A very similar but often less severe disease is caused by Salmonella serotype paratyphi. Both Typhoid and paratyphoid A and B fever are referred to as enteric Fevers. Typhoid fever is a life-threatening illness which needs early identification and appropriate treatment to avoid serious complications.

Cause

Typhoid and paratyphoid fever are caused by Bacilli Salmonella typhi and salmonella paratypi respectively

Epidemiology

Typhoid fever is more common in areas of the world where hand washing is less frequent and water is likely to be contaminated with sewage .In the United States about 400 cases occur each year, and 75% of these are acquired while travelling internationally. Typhoid fever is still common in the developing world, where it affects about 21.5 million persons each year.

In Uganda, more than 80 percent of the districts continue to report cases of typhoid fever to the Ministry of Health. With improved reporting from the districts to the MoH, more cases and deaths due to typhoid fever are reported on a weekly basis. In the period January to December 2001, a total number of 2,101 cases and 7 deaths due to typhoid fever were reported to the Epidemiological Surveillance Division. The corresponding number of cases and deaths reported during 2002 were 7,397 and 21 respectively. These reports were regularly received from 48 out of the 56 districts (86%).

The trend over 24 months seems to suggest a general increase in morbidity due to typhoid fever in the country. The geographical distribution indicates that 12 districts located in central and western parts of the country are most affected. The mortality due to typhoid fever in Uganda is very low (CFR=0.3%). However, available research indicates that even where there is ample treatment, the case-fatality rate for typhoid is high (10-20%). This indicates that typhoid fever is a silent epidemic in the country.

- Transmission, disease process and incubation period
- Clinical presentation and diagnosis

Transmission
Salmonella Typhi lives only in humans. Persons with typhoid fever carry the bacteria in their bloodstream and intestinal tract. In addition, a small number of persons, called carriers, recover from typhoid fever but continue to carry the bacteria. Both ill persons and carriers shed S. Typhi in their feces (stool).

Typhoid fever is spread by eating food or drinking beverages that have been handled by a person who is shedding Salmonella typhi or if sewage contaminated with Salmonella typhi gets into the water used for drinking or washing food.

Disease process
After ingestion in food or water, typhoid organisms pass through the pylorus and reach the small intestine. They rapidly penetrate the mucosal epithelium, where they rapidly educe an influx of macrophages that ingest the bacilli but do not generally kill them. Some bacilli remain within macrophages of the small intestinal lymphoid tissue. During an acute infection, S. typhi multiplies in phagocytic cells before being released into the bloodstream. Other typhoid bacilli are drained into mesenteric lymph nodes where there is further multiplication and ingestion macrophages. It is believed that typhoid bacilli reach the bloodstream principally by lymph drainage from mesenteric nodes, after which they enter the thoracic duct and then the general circulation. As a result of this silent primary bacteraemia the pathogen reaches an intracellular haven within 24 hours after ingestion throughout the organs of the reticulo-endothelial system that is spleen, liver, bone marrow.

Incubation Period
Usually the incubation period ranges from 8 to 14 days however it can be as short as 3 days to as long as 60 days depending on quantity of inoculums, and on host factors such as immunity.

Clinical presentation
Many factors influence the severity and overall clinical outcome of the infection. They include the duration of illness before the initiation of appropriate therapy, the choice of antimicrobial treatment, age, the previous exposure or vaccination history, the virulence of the bacterial strain, the quantity of inoculums ingested, host factors (e.g. AIDS or other immunosuppressant) and whether the individual was taking other medications such as Histamine blockers or antacids to diminish gastric acid. Patients who are infected with HIV are at significantly increased risk of clinical infection with

S. typhi and S. paratyphi .Evidence of Helicobacter pylori infection also represents an increased risk of acquiring typhoid fever.

Due to the above stated factors the clinical presentation of typhoid fever depends on an individual. It varies from a mild illness with low-grade fever, malaise, and slight dry cough to a severe clinical picture with abdominal discomfort and multiple complications.

There are three forms of clinical presentation:
Acute non-complicated disease:
Complicated disease:
Carrier state

Acute non-complicated disease
Acute typhoid fever is characterized by prolonged fever and up to 25% of patients show exanthema (rose spots), on the chest, abdomen and back. The temperature rise in a step like pattern. There are also disturbances of bowel function such as constipation in adults and diarrhoea in children. Adults especially in the second week of infection may also develop diarrhoea characteristic of pea soup colour. Other symptoms include headache, malaise and anorexia. Bronchitic cough is also common in the early stage of the illness.

Complicated disease
Acute typhoid fever may be severe. Depending on the clinical setting and the quality of available medical care, up to 10% of typhoid patients may develop serious complications. Since the gut-associated lymphoid tissue exhibits prominent pathology, the presence of occult blood is a common finding in the stool of 10-20% of patients, and up to 3% may have melena.

Intestinal perforation has also been reported in up to 3% of hospitalized cases. Abdominal
discomfort develops and increases. It is often restricted to the right lower quadrant but may be diffuse. This may be followed by symptoms and signs of intestinal perforation and peritonitis accompanied by a sudden rise in pulse rate, hypotension, marked abdominal tenderness, rebound tenderness and guarding, and subsequent abdominal rigidity.

Altered mental status in typhoid patients has been associated with a high case-fatality rate. Such patients generally have delirium, rarely with coma. Typhoid meningitis, encephalomyelitis, Guillain-BarrÃ syndrome, cranial or peripheral neuritis,
and psychotic symptoms, although rare may occur.

Other serious complications due to typhoid fever include haemorrhages (causing rapid
death in some patients), hepatitis, myocarditis, pneumonia, disseminated

intravascular coagulation, thrombocytopenia and hemolyticuraemic syndrome.

Carrier state
About 1-5% of patients, depending on age, may harbour S.typhi in the gallbladder hence becoming chronic carriers. Carriers do not show any sign but continue shedding the salmonella typhi in the stool and can transmit the infection.

Case definition

Confirmed case of typhoid fever; A patient with fever (38Â°C and above) that has lasted for at least three days, with a laboratory-confirmed positive culture (blood, bone marrow, bowel fluid) of S. typhi.

Probable case of typhoid fever; A patient with fever (38Â°C and above) that has lasted for at least three days, with a positive serodiagnosis or antigen detection test but without S. typhi isolation.

Chronic carrier; This is a person who excretes S. typhi in stools or urine (or has repeated positive bile or duodenal string cultures) for longer than one year after the onset of acute typhoid fever. Short-term carriers also exist but their epidemiological role is not as important as that of chronic carriers. Some patients excreting S. typhi have no history of typhoid fever.
The rate of carriage is slightly higher among female patients, older than 50 years, and patients with cholelithiasis or schistosomiasis

Diagnosis
The definitive diagnosis of typhoid fever depends on the isolation of S. typhi from blood, bone marrow or a specific anatomical lesion. The presence of clinical symptoms characteristic of typhoid fever or the detection of a specific antibody response by widal test is suggestive of typhoid fever but not definitive.

A culture can be done to confirm the diagnosis. A sample of blood, Stool or rectal swab can be taken off for this purpose.

Management of typhoid fever

General management
Supportive measures are important in the management of typhoid fever, such as oral or intravenous hydration, the use of antipyretics, and appropriate nutrition and blood transfusions if indicated. More than 90% of patients can be managed at home with oral antibiotics, reliable care and close medical follow-up for complications or failure to respond to therapy. However, patients with persistent vomiting, severe diarrhea and abdominal distension may require hospitalization and parenteral antibiotic therapy.

Management of uncomplicated typhoid fever

Antimicrobial therapy

There is a wide range of antibiotics used in treatment of typhoid,Due to this fact together with the emergence of multidrug resistance knowledge of the antibiotic sensitivity of the infecting strain is crucial in determining drug choice. If no culture is available a knowledge of likely sensitivity as indicated by the available global data may be useful

Efficacy, availability and cost are important criteria for the selection of first-line antibiotics to be used in developing countries. The therapeutic strategies for children, e.g. the choice of antibiotics, the dosage regimen and the duration of therapy, differ from those for adults.

Chloramphenicol, despite the risk of agranulocytosis in 1 per 10 000 patients,is still widely prescribed in developing countries for the treatment of typhoid fever. S.Typhi remains sensitive to this drug and it is widely available in most primary care settings .The disadvantages of using chloramphenicol include a relatively high rate of relapse (5- 7%), long treatment courses (14 days) and the frequent development of a carrier state in adults.

Dose:

The recommended dosage is 50-75 mg per kg per day for 14 days in four divided doses. The usual adult dose is 500 mg given four times a day for 14 days.

Ampicillin and amoxicillin may also be used at 50 to 100 mg per kg per day orally, intramuscularly or intravenously divided into three or four doses. No benefit has been reported to result from the addition of clavulanic acid to amoxicillin.

Trimethoprim-sulfamethoxazole, (TMP-SMZ) can be used orally or i.v. in adults at a dose of 160 mg TMP plus 800 mg SMZ twice daily or in children at 4 mg TMP per kg and 20 mg SMZ per kg for 14 days (55).

Fluoroquinolones are also used in the treatment of typhoid. They are widely regarded as optimal for the treatment of typhoid fever in adults. They include drugs like ofloxacin, ciprofloxacin, fleroxacin, perfloxacin. They are relatively inexpensive, well tolerated and more rapidly and reliably effective than the former first-line drugs, viz. chloramphenicol, ampicillin,amoxicillin and trimethoprim-sulfamethoxazole. The majority of isolates are still sensitive. This so because the optimum treatment for quinolone-resistant typhoid fever has not been determined.Azithromycin,the third-generation cephalosporins, or a

10-14 day course of high-dose fluoroquinolones, is effective.

Management of complicated typhoid fever

Both outpatients and inpatients with typhoid fever should be closely monitored for the development of complications.

In severe typhoid the fluoroquinolones are given for a minimum of 10 days.

High-dose of intravenous dexamethasone in addition to antimicrobials can be given if the findings are normal and typhoid meningitis is suspected.

Dose:

Initial dose of dexamethasone is 3 mg/kg by slow i.v. infusion over 30 minutes and after six hours, 1 mg/kg is administered and subsequently repeated at six-hourly intervals to lower the mortality rate.

Patients with mental disturbance should check for meningitis.

Patients with intestinal haemorrhage need intensive care, monitoring and blood transfusion.

In case of intestinal perforation, surgical repair should not be delayed longer than six hours. Metronidazole and gentamicin or ceftriazone should be administered before and after surgery (if a fluoroquinolone is not used) to treat leakage of intestinal bacteria into the abdominal cavity.

Management of carriers

Relapses involving acute illness occur in 5-20% of typhoid fever cases that have apparently been treated successfully. A relapse is characterized by the return of fever soon after the completion of antibiotic treatment the clinical manifestation is frequently
milder than the initial illness. At this stage culture is a must and schistosomiasis should be ruled out.

If cholelithiasis or schistosomiasis is present cholecystectomy is done, antiparasitic medication in addition to antibiotics are given in order to eradicate S. typhi carriage

Amoxicillin or ampicillin (100 mg per kg per day) plus probenecid (BenemidÂ®) (1g orally or 23 mg per kg forchildren)or TMP-SMZ (160 to 800 mg twice daily) is administered for six weeks; about 60% of persons treated with either regimen can be expected to have negative cultures on follow-up.

Clearance of up to 80% of chronic carriers can be achieved with the administration of 750 mg of ciprofloxacin twice daily for 28 days or 400 mg of norfloxacin. Other quinolone drugs may yield similar results.

Effects of typhoid on the government and community:

Increased expenditure on treating the infected people.
Reduction on trade as business men may fear to go to affected area
Increased Morbidity leading to absentism from work thus reduced production
Increased Mortality leading to psychological trauma, increased orphan hood and other related effects
Poverty due to increased morbidity and medical expenses
Stigmatization as typhoid is attached to poor sanitation.

Prevention and control

Typhoid fever can be prevented by the following measures:

Avoiding the risky foods and drinks

Being conscious of what one eats and drinks may be as important as being vaccinated. This is because the vaccines are not completely effective. Avoiding risky foods is a good practice because it also protects an individual from other illnesses, including diarrhea, cholera, dysentery, and hepatitis A.
Drinking water should be boiled cooled and kept covered in clean containers. Only carbonated bottled water as opposed to uncarbonated should be taken because it is safer. If one wants to take chew Ice it should be made from either boiled or bottled water
Foods should be thoroughly cooked and served hot and steaming. Food handlers should be screened for typhoid
Raw vegetables and fruits should be washed thoroughly and peeled before eating. It is advised that someone should peel his/her own fruit to reduce the risk of contamination. Hand washing with soap and water must be done prior to peeling the fruits and the peelings should not be eaten.
Foods and beverages from street vendors should be avoided as it is difficult for food to be kept clean on the street, and many people get sick from food bought from street vendors.
Mothers not living with HIV should breastfeed for two years to protect their children from risky foods and also to provide immunity
The public should be encouraged to always to remember a slogan "Boil it, cook it, peel it, or forget it" if they are to avoid risky food that can predispose them to typhoid.

Immunisation

Immunisation has little value however it is usually recommended to people

travelling to countries where typhoid is common. Typhoid vaccines lose effectiveness after several years therefore booster doses are recommended after a given period

Below is a table showing the basic information on typhoid vaccines.

Vaccine Name	How given	Number of doses necessary	Time between doses	Total time needed to set aside for vaccination	Minimum age for vaccination	Booster needed every...
Ty21a (VivotifBerna, Swiss Serum and Vaccine Institute)	1 capsule by mouth	4	2 days	2 weeks	6 years	5 years
ViCPS (Typhim Vi, Pasteur Merieux)	Injection	1	None	2 weeks	2 years	2 years

Use of multi-sectoral approach

Typhoid cannot be prevented and controlled by only the health sector therefore a comprehensive multidisciplinary approach should be adopted. The sectors of importance in typhoid control include those providing safe water and maintaining good sanitation for example the national water and sewerage co-operation of Uganda, those involved in Education and communication such as the ministry of Education.

The sector responsible for providing water should put in place the following control measures.

In urban areas, control and treatment of the water supply systems must be strengthened from catchments to consumer. Safe drinking water should be made available to the population through a piped system or from tanker trucks.
In rural areas, wells must be checked for pathogens and treated if necessary. At home, a particular attention must be paid to the disinfection and the storage of the water however safe its source. Drinking-water can be made safe by boiling it for one minute or by adding a chlorine-releasing chemical. Narrow-mouthed pots with covers for storing water are helpful in reducing secondary transmission of typhoid fever. Chlorine is ineffective when water is stored in metallic containers.

In some situations, such as poor rural areas in developing countries or refugee camps, fuel for boiling water and storage containers may have to be supplied

The sector responsible for sanitation should ensure the following:
Availing the appropriate facilities for human waste disposal to all community.
Building pit latrines in an emergency.
Collecting and treating sewage, especially during the rainy season.
Discouraging the use human excreta as fertilizers

Political and community Involvement

The policy makers and leaders should make appropriate policies to implement the suggested measures for example screening and issuing licenses to all the food handlers.
Substantial resources must be allocated to institution supporting the preventive measures.
The community leaders must be involved in educating their people about the importance of good sanitation and preventive measures of typhoid

Recommendations

Since typhoid appears to be silent epidemic in most of the developing recommendations are made;

1.Every person should be responsible enough to take precautions to prevent the spread and the acquisition of typhoid.
2.The convalescent patients and carrier should be reviewed by health workers before they resume on duty more especially food handlers.
3.All government ministries should work together to prevent typhoid since the ministry of health it is not the only player in the control of Typhoid fever.
4.The government should put in place policies supporting the prevention of typhoid such as screening all food handlers.
5.The government should allocate reasonable resources to support the public health intervention focusing on preventing communicable diseases.
6.Health facilities should be well stocked with drugs and other utilities to effectively treat the sick.

Conclusion

Typhoid fever is common in developing countries especially where handwashing is rarely practised. Poor sanitation contributes a lot to the high typhoid prevalence rate in the such countries. Though it is easily spread it can be prevented and completely treated. It is therefore a responsibility of both the public and government to be vigilant to prevent its spread and also to treat the infected in order to reduce both the high mortality and morbidity rate due to Typhoid fever.

BILHARZIA (or schistosomias)

Introduction

Bilharzia or schistosomiasis (also called 'snail fever') is a parasitic disease caused by several species of flukes, schistosoma. Schistosomiasis remains one of the most prevalent helminthic infections in the world. It is commonly found in tropical and subtropical areas of Africa, South America, Middle East, and East Asia; especially in areas with water contaminated with freshwater snails, which may carry the parasite. The disease affects many people by wading or swimming in lakes, ponds and other bodies of water. Although it has a low mortality rate, 'bilharzia or bilharziosis' is a chronic illness associated with renal and bladder dysfunction.

- **Types**

  There are five species of flatworms that cause Schistosomiasis namely; S. mansoni, S. haematobium, S. intercalatum, S. japonicum, S. mekongi. Each causes a different clinical presentation of the disease (see table 1). Schistosomiasis may localize in different parts of the body, and its localization determines its particular clinical profile.
  The geographical distribution of the different Schistosoma depends on: distribution of the distinct snail species that serve as intermediate hosts, climate and ecological factors that regulate the snail population, patterns of water supply, quality, distribution and human use.

  Table:1 Geographical distribution and epidemiology

Species	Geographical distribution	Vector snails	Disease
S. mansoni	South America,Caribbean, Africa and Middle East	Biomphalaria	intestinal schistosomiasis
S. haematobium	Africa,Middle& Far East	Bulinus	urinary schistosomiasis
S. intercalatum	Central and West Africa		intestinal schistosomiasis
S. japonicum	Far East	Oncomelania	intestinal schistosomiasis
S. mekongi	Mekong river of Southeast Asia		intestinal schistosomiasis

Life cycle

Schistosoma have a typical trematode vertebrate-invertebrate lifecycle, with humans being the definitive host. Parasite eggs are released into the environment from infected individuals, hatching on contact with fresh water to release the free-swimming miracidium. Miracidia infect fresh-water snails by penetrating the snail's foot.

After infection, close to the site of penetration, the miracidia transforms into germs and new parasites, known as cercariae (larvae), which can infect human beings. The newly transformed schistosomulum may remain in the skin for 1-2 days before locating a post-capillary venule. Eight to ten days after penetration of the skin, the parasite migrates to the liver sinusoids.
Parasites reach maturity in 6-8 weeks, at which time they begin to produce eggs. Many of the eggs pass through the walls of the blood vessels, and through the intestinal wall, to be passed out of the body in faeces.
Trapped eggs mature normally, secreting antigens (a substance that prompts the generation of antibodies; proteins that are found in blood or other bodily fluids, and are used by the immune system to identify and neutralize foreign bodies). The eggs themselves do not damage the body. Rather it is the cellular infiltration resultant from the immune response that causes the pathology classically associated with schistosomiasis.

Clinical features
Schistosomiasis is a chronic disease. Acute schistosomiasis may occur weeks after initial infection by S. mansoni and or S. japonicum. Manifestations develop between 4 and 8 weeks after exposure. They include: fever, sweat chills, cough and headaches.

A patient with intestinal Schistosomiasis may complain of abdominal pain, fatigue, weakness in leg, bowel, stomach muscles, bloody or diarrhoea, eosinophilia (extremely high eosinophil granulocyte count). Ulceration and chronic bleeding may lead to moderate or severe iron deficiency (anaemia). Chronic schistosomiasis is a less dramatic and progressive illness resulting from prolonged tissue injury and severe organ damage of hepatosplenomegaly (enlargement of both the liver and the spleen).
Urinary Schistosomiasis usually creates bladder and compromised kidney function, haematuria, bladder cancer, and uretitis.
Pulmonary Schistosomiasis results from parasite egg deposition in walls of blood vessels obstructing blood circulation resulting into pulmonary hypertension.

Occasionally central nervous system (CNS) lesions, embolic egg granulomas in brain or spinal cord (CNS Schistosomiasis) occur with S. mansoni, S. japonicum infection in chronically infected persons but may happen during the acute phase.

Diagnosis of disease

The gold standard for Schistosoma infection is identification of parasite eggs in patient stool (S. mansoni, S. japonicum, S. mekongi and S. intercalatum) or urine (S. haematobium).
Tissue biopsy (rectal biopsy for all species and biopsy of the bladder for S. haematobium) may demonstrate parasite infection when stool or urine examinations are negative.
Serology or antibody detection can be used to detect presence of specific antischistosome antibodies.

Treatment

Bilharzia, if diagnosed early can be treated using a single oral dose of the drug Praziquantel annually. Cure rates have typically been equal to or greater than 85%. Worm pairs can live in the body for an average of four and a half years, but may persist up to 20 years.
In younger patients, completion of therapy is associated with reduction, regression or reversal of complaints. Older people with more advanced tissue injury may not be able to experience reversal of their schistosome-associated lesions.

Prevention and Control

There are a number of different ways to prevent transmission of infection or reduce the likelihood of heavy infection. Community treatment of Schistosomiasis may be based on the impact of the disease.

For example, in endemic villages:
When >50% of children have blood in their urine, everyone in the village receives treatment.
When 20% to 50% of children have bloody urine, only school-age children are treated.
When less than 20% of children have symptoms, mass treatment is not implemented.

Prevention is best accomplished by:

1)Reduction or elimination of water-dwelling snails which are the natural reservoir of
the disease.
2) Elimination or guard against snail habitats or likely to harbour the carrier snails.
3)Sanitation and hygiene measures to prevent human excreta from contaminating local
water sources
4)Use of protective footwear or clothing
5)Treatment of exposed populations

HYPERTENTION

Definition of hypertension

Hypertension is the persistent high blood pressure, in which the systolic is more than 140mm of Hg, and diastolic is more then 90mm of Hg (140/90mm Hg) which are measured in two or more consecutive days in a week. It can also described as high blood pressure (tension) in the arteries (vessels that carry blood from the pumping heart to all the tissues and organs of the body).

The top number, the systolic blood pressure, corresponds to the pressure in the arteries as the heart contracts and pumps blood forward into the arteries. The bottom number, the diastolic pressure, represents the pressure in the arteries as the heart relaxes after the contraction. The diastolic pressure reflects the lowest pressure to which the arteries are exposed.

Epidemiology of hypertension

The prevalence of hypertension appears to vary depending on geographical area, socio-economic status, age of persons, gender, heredity, and ethnicity.

Epidemiological studies show that there are significant geographical differences in the occurrence of hypertension and its complications both between and within countries; this is considered to be influenced by the interaction of nutritional and environmental factors with the subject's genetic predisposition/susceptibility to develop hypertension. The American Heart Association estimates high blood pressure affects approximately one in three adults in the United States - 73 million people. High blood pressure is also estimated to affect about two million American teens and children, and the Journal of the American Medical Association reports that many are under-diagnosed.

In developed countries with affluent economies, higher levels of blood pressure and higher prevalence of hypertension in lower socio-economic groups have been noted; this inverse relation has been also noted with levels of education, income and occupation. In societies that are in the transitional stage of economic and epidemiological change, higher levels of blood pressure and a

higher prevalence of hypertension have been noted in upper socio-economic groups.

Mean blood pressure and prevalence of hypertension increase with age throughout childhood, adolescence and adulthood in most populations of developed and developing countries. However, in some isolated populations, this age-related rise of blood pressure (BP) is not evident.

With regard to gender, men tend to display higher blood pressure than women, more evident in youth and middle-age. Later in life (over 50 years old), the difference narrows and the pattern may be reversed.
Although the precise mode of heredity/inheritance has not yet been demonstrated, a high occurrence of hypertension is observed among subjects with a family history of hypertension and it is higher and more severe when both parents are concerned.

Studies have also revealed higher blood pressure levels in the black community than in other ethnic groups, mainly in black Afro-Americans with early onset, severity and appearance of complications.

Mortality in African adults is unknown and probably varies considerably between regions from 1% to 2.5% per year. Community and hospital studies suggest that 5% to 15% of people die from cardiovascular diseases mainly stroke and congestive heart failure resulting from hypertension. Calculation of the population attributable risk confirms that about 5% of deaths can be attributed to hypertension. Given that half of all deaths occur in adults, the overall contribution of hypertension would therefore be around 2.5%. One study of global disease burden has attributed 5.8% of deaths at all ages to hypertension.

Types of hypertension

There are two major type of hypertension, namely:
1. Essential hypertension (primary hypertension)
This accounts for 95% of all hypertension cases. The cause of essential hypertension is multifactorial, that is, there are several factors whose combined effects cause this (e.g. genetic factors, lifestyle). Approximately 30 % of cases of essential hypertension are said to be attributable to genetic factors. People who suffer from essential hypertension have an increased resistance (i.e. stiffness or lack of elasticity) in the tiny arteries that are most distant from heart.

2. Non essential hypertension (secondary hypertension)
This accounts for 5% of all hypertension cases. This type of hypertension is secondary to (caused by) a specific abnormality in one of the organs or systems

of the body.
These two major types can further be classified as below:

- Accelerated hypertension, which is the progressive hypertension with the funduscopic vascular changes.
- Adrenal hypertension, which is associated with an adrenal tumor that secretes excess mineral corticoids (hormones produced by the adrenal glands)
- Borderline hypertension. in which the arterial blood pressure is sometimes within the normal range and sometimes within the hypertensive range, thus fluctuating between the normal and the abnormal.
- Gestational hypertension, developed by pregnant mothers.
- Malignant hypertension. This is a severe hypertensive state with papilledema of the ocular fundus and vascular hemorrhagic lesions, thickening of the small arteries and arterioles, left ventricular hypertrophy, and poor prognosis.
- Ocular hypertension. This develops in the intra ocular region, without signs of glaucoma (increased pressure in the eye)
- Portal hypertension, which is abnormally increased pressure in the portal veins which brings blood to the liver. This is a common complication with liver cirrhosis
- Renal hypertension, which is associated with or due to renal disease
- Pulmonary hypertension, which is the pressure exerted in the pulmonary arteries. It is associated with chronic obstructive respiratory diseases (e.g. asthma, bronchitis)
- Systemic venous hypertension. This is characterised by the elevation of systemic venous pressure, usually detected by inspection of the jugular veins.

Risk factors for hypertension
In Africa, as elsewhere, obesity and sodium intake are risk factors for hypertension. In industrialised societies such as the United States, obesity accounts for 25% of cases of hypertension. However, the relative leanness of Africans means that the contribution of obesity to high blood pressure is only around 10%, though this figure may be going up given the westernisation and urbanization of living styles in vogue.

Causes of essential hypertension
The actual causes of essential hypertension, its basic causes or underlying defects are not always known. Nevertheless, certain associations have been recognized in people with essential hypertension.

- Essential hypertension develops only in groups or societies that have a fairly high intake of salt, exceeding 5.8 grams daily, especially in the hypertension that is associated with advancing age, African American

background, obesity, hereditary (genetic) susceptibility, and kidney failure (renal insufficiency). Excess salt intake causes an increase in retention of fluids in the body, thereby putting excessive workload on the heart and the kidney systems to remove the excess fluids. The result is high blood pressure.

- Genetic factors are also thought to play a prominent role in the development of essential hypertension. Approximately 30% of cases of essential hypertension are attributable to genetic factors. However, the genes for hypertension have not yet been identified. The responsible genes are believed to regulate blood pressure by controlling salt balance and the tone (state of elasticity) of the arteries. Genetic factors are said to contribute about 30% of cases of essential hypertension. Also, in individuals who have one or two parents with hypertension, high blood pressure is twice as common as in the general population.
- Vascular diseases, such as arteriosclerosis (occlusion of the blood vessels). This condition causes an increased resistance (stiffness or lack of elasticity) in the tiny arteries that are most distant from the heart (peripheral arteries or arterioles). This condition is also present in those individuals whose essential hypertension is associated with genetic factors, obesity, lack of exercise, overuse of salt, and ageing.

Causes of secondary hypertension

- *Diseases of the kidneys* can cause secondary hypertension. This can be due to narrowing (stenosis) of the artery that supplies blood to the kidneys (renal artery). In younger individuals, usually women, the narrowing is caused by a thickening of the muscular wall of the arteries, while in older individuals, it is due to hard, fat-containing (atherosclerotic) plaques that are blocking the renal artery. Any of the other types of chronic kidney disease that reduces the function of the kidneys can also cause hypertension due to hormonal disturbances and/or retention of salt.
- *Adrenal gland tumours*. The adrenal glands sit right on top of the kidneys. When the glands develop tumours, the latter produce excessive amounts of adrenal hormones that cause high blood pressure.
- *Coarctation of the aorta*, a rare hereditary disorder, is one of the most common causes of hypertension in children. This condition is characterized by a narrowing of a segment of the aorta, the main large artery coming from the heart. The narrowed segment (coarctation) of the aorta generally occurs above the renal arteries, which causes a reduced blood flow to the kidneys thereby stimulating the kidneys to produce renin-angiotensin-aldosterone hormone which elevates the blood pressure.
- *Obesity*. This is a genetic or acquired body morphological condition whereby ones BMI (body mass index) index â‰¥ 25. It is characterised by excessive fat deposits that may occlude the blood vessels thereby

increasing resistance to blood flow through the vessels. The result is high blood pressure or hypertension.
- *Sedentary lifestyle* is one the leading causes of obesity, which eventually leads to hypertension as illustrated above.

Signs, symptoms and complications of hypertension
Uncomplicated high blood pressure usually occurs without any symptoms for years (silently) until when one develops fatal complications, hence labelled "the silent killer.□ This happens when there are no symptoms, and those affected fail to undergo periodic blood pressure screening. However, the common symptoms are:

- dizziness
- headache
- night sweating
- nausea
- hearing own heart sound (palpitation)
- blurred vision
- shortness of breath
- blood pressure above 140/90mm of Hg

In severe high blood pressure (accelerated or malignant hypertension) all the above symptoms are present, but more severe with the diastolic blood pressure (the minimum pressure) exceeding 140 mm Hg. Malignant hypertension is a medical emergency and requires urgent treatment to prevent a stroke (brain damage).

While elevated blood pressure alone is not an illness, it often requires treatment due to its short- and long-term effects on many organs. The common complications include:

- hypertensive cardiomyopathy leading to heart failure
- renal damage (kidney failure)
- cerebrovascular accident (stroke)
- retinal damage (retinopathy)
- myocardial infraction (heart attack)

Diagnosis of hypertension
Diagnosis of hypertension is generally on the basis of a persistently high blood pressure. Usually this requires three separate measurements at least one week apart. Exceptionally, if the elevation is extreme, or end-organ damage is present then the diagnosis may be applied and treatment commenced immediately.

Obtaining reliable blood pressure measurements relies on following strict guidelines and procedures, validating diagnosis methods, and understanding

the many factors that influence blood pressure reading. For instance, measurements in control of hypertension should be at least 1 hour after caffeine, 30 minutes after smoking or strenuous exercise and without any stress. Cuff size is also important. The bladder should encircle and cover two-thirds of the length of the (upper) arm. The patient should be sitting upright in a chair with both feet flat on the floor for a minimum of five minutes prior to taking a reading. The patient should not be on any adrenergic stimulants, such as those found in many cold medications.

Some investigations done in diagnosed or suspected hypertensive clients to identify any underlying cause or detect any damage already done include:

- blood tests (creatinine, electrolytes, cholesterol, glucose)
- urine analysis for proteinuria (for kidney or renal damage especially among the diabetics)
- electrocardiogram (for evidence of the heart being under strain from working under a high blood pressure)
- chest x-ray (to check for any signs of cardiac enlargement or disorder)

Control and treatment of hypertension
The presence of symptoms can be a good thing in that they can prompt people to consult a doctor for treatment and make them more compliant in taking their medications. Often, however, a person's first contact with a physician may be after significant damage to the end-organs has occurred. In many cases, a person visits or is brought to the doctor or an emergency room with a heart attack, stroke, kidney failure, or impaired vision (due to damage to the back part of the retina).

In order to prevent and control the occurrence of hypertension and its complications, the following measures should be taken:

- Public awareness on the causes and effects of hypertension
- Frequent blood pressure screening should be done to identify patients with undiagnosed high blood pressure before significant complications have developed.
- Self regulation of salt intake should be practised. Adults above 19 years of age should consume less than 5 grams of salt per day (to replace the average amount lost daily through perspiration and to achieve a diet that provides sufficient amounts of other essential nutrients).
- Life style modification. Smoking, excessive alcohol intake should be avoided. Lifestyles that lead to obesity such as eating fast foods, and lack of exercises should be also be avoided. A DASH diet (dietary approaches to stop hypertension), which is rich in fruits and vegetables and low fat or fat-free dairy foods has been found by the US National Institutes of Health beneficial in avoidance and control of hypertension.

- Stressful situations such as high sound levels or high illuminations should not be entertained. People should always seek personal services of counsellors or use other stress relieving interventions like meditation and prayer.
- People already diagnosed with hypertension should adhere to the prescribed anti hypertensive drugs to avoid developing complications. Commonly used drugs are:
    - angiotensin converting enzyme inhibitors : such as captopril, enalpril
    - angiotensin receptor antagonist : such as irbesartan,
    - alpa blockers, e.g. prazosin
    - beta blockers, e.g. metoprolol, atenolol, labetalolol.
    - calcium channel blockers, e.g. amlodipine verapamil
    - diuretics: hydrochlorothiazide
    - direct rennin inhibiter: teckturna
- Surgery can be done to correct an associated abnormality. For example, a narrowing of the renal artery may be treated by balloon angioplasty (widening of the renal artery or dilating of the coarctation of aorta) Tumours in the adrenal gland can also be removed in the treatment of malignant hypertension.

Challenges in prevention, control and treatment

- In sub-Saharan Africa it is difficult to formulate and justify policy on treating chronic conditions such as hypertension as there are no health statistics from which to judge likely costs and benefits
- The majority of Africans live in rural areas and are marginally integrated into the cash economy, while some of those who live cities are in extreme poverty In this case, the challenge lies in developing effective strategies for these sections of society.
- Hypertension is the most common cardiovascular condition in the world and the problem of defining a strategy for control confronts all societies. Hypertension is fully treatable, but social conditions in Africa make the implementation of blood pressure control programmes difficult. Lack of a clear strategy based on evidence has undermined further these efforts.
- High blood pressure is a silent killer in that initially it may cause no symptoms but can still cause serious long-term complications. Many people have high blood pressure and don't even know it.
- Psychosocial factors in hypertension have been studied little. In Africa Instruments for measuring these factors in African societies have not been much developed. No trials of preventive measures that have reduced risk factors for hypertension have been reported from most of Africa. Drug treatment is therefore at the moment the only proved option at present.

Emerging issues regarding hypertension

1. It is important to remember that not only can kidney disease cause hypertension, but hypertension can also cause kidney disease. Therefore, all patients with high blood pressure should be evaluated for the presence of kidney disease so they can be treated appropriately.
2. Control of iodine deficiency disorders using salt versus salt intake being one of the risk factors for the development of hypertension

Conclusion
High Blood Pressure (Hypertension) at a glance:

- Essential hypertension may run in some families and occurs more often in the African American population, although the genes for essential hypertension have not yet been identified.
- Though hypertension may run in some families (genetic), high salt intake, obesity, lack of regular exercise, excessive alcohol or coffee intake, and smoking may all adversely affect the outlook for the health of an individual with hypertension. Lifestyle adjustments in diet and exercise and compliance with medication regimes are important factors in determining the outcome for people with hypertension.
- High blood pressure is called "the silent killer" because it often causes no symptoms for many years, even decades, until it finally damages certain critical organs.
- Poorly controlled hypertension ultimately can cause damage to blood vessels in the eye, thickening of the heart muscle and heart attacks, hardening of the arteries (arteriosclerosis), kidney failure, and strokes.
- Heightened public awareness and screening of the population are necessary to detect hypertension early enough so it can be treated before critical organs are damaged.

DIPTHERIA

Definition of diphtheria
Diphtheria is a disorder caused by a highly contagious bacterial infection called bacterium Corynebacteriumdiphtheriae. Diphtheria spreads easily and occurs quickly. It mainly affects the nose and throat. Children under 5 and adults over 60 years old are particularly at risk for contracting the infection. People living in crowded or unclean conditions, those who aren’t well nourished, and children and adults who don't have up-to-date immunizations are also at risk.

Description of diphtheria
Diphtheria bacteria live in an infected person's nose, throat, skin, or eye

discharges. It is especially dangerous when it affects the throat, where it can produce a thick grey membrane that may grow large enough to obstruct breathing. The most serious complications are caused by a toxin produced by the diphtheria bacterium that can damage the heart, nervous system and, less often, the kidneys.

Diphtheria is highly contagious. It's easily passed from the infected person to others through sneezing, coughing, or even laughing. It can also be spread to others who pick up tissues or drinking glasses that have been used by the infected person. You can also catch diphtheria from touching the open sores on someone with skin diphtheria.

People who have been infected by the diphtheria bacteria can infect others for up to 4 weeks, even if they don't have any symptoms. The incubation period (the time it takes for a person to become infected after being exposed) for diphtheria is 2 to 4 days, although it can range from 1 to 6 days.

Signs and symptoms

In its early stages, diphtheria can be mistaken for a bad sore throat. A low-grade fever and swollen neck glands are the other early symptoms.

The toxin, or poison, caused by the bacteria can lead to a thick coating in the nose, throat, or airway. This coating is usually fuzzy gray or black and can cause breathing problems and difficulty in swallowing. The formation of this coating (or membrane) in the nose, throat, or airway makes a diphtheria infection different from other more common infections (such as strep throat) that cause sore throat.

As the infection progresses, the person may have difficulty breathing or swallowing, complain of double vision, have slurred speech, even show signs of going into shock (skin that's pale and cold, rapid heartbeat, sweating, and an anxious appearance).

In cases that progress beyond a throat infection, diphtheria toxin spreads through the bloodstream and can lead to potentially life-threatening complications that affect other organs of the body, such as the heart and kidneys. The toxin can cause damage to the heart that affects its ability to pump blood or the kidneys' ability to clear wastes. It can also cause nerve damage, eventually leading to paralysis. Up to 40% to 50% of those who don't get treated can die.

Prevention and treatment

Preventing diphtheria depends almost completely on immunising children with the diphtheria/tetanus/pertussis (DTP or DTaP) vaccine and non-immunized adults with the diphtheria/tetanus vaccine (DT). Most cases of diphtheria occur in people who haven't received the vaccine at all or haven't received the entire course.

The immunisation schedule calls for:

- DTaP vaccines at 2, 4, and 6 months of age
- Booster dose given at 12 to 18 months
- Booster dose given again at 4 to 6 years
- Booster shots given every 10 years after that to maintain protection

Although most children tolerate it well, the vaccine sometimes causes mild side effects such as redness or tenderness at the injection site, a low-grade fever, or general fussiness or crankiness. Severe complications, such as an allergic reaction, are rare. If the infection is advanced, people with diphtheria may need a ventilator to help them breathe. In cases in which the toxins may have spread to the heart, kidneys, or central nervous system, patients may need intravenous fluids, oxygen, or heart medications. A person with diphtheria must also be isolated. Family members (as well as others who spend a lot of time with the person with diphtheria) who haven't been immunized, or who are very young or elderly, must be protected from contact with the patient. When someone is diagnosed with diphtheria, it is necessary to treat everyone in the household who may have been exposed to the bacteria. This will include assessment of immune status, throat cultures, and booster doses of the diphtheria vaccine. They will also receive antibiotics as a precaution.

Immediate hospitalisation and early intervention allow most patients to recover from diphtheria. After the antibiotics and anti-toxin have taken effect, someone with diphtheria will need bed rest for a while (4 to 6 weeks, or until full recovery). Bed rest is particularly important if the disease has affected the person heart.

In conclusion, there is need to encourage immunisation in early childhood days as well as ensuring proper sanitation of our surroundings. Immediate treatment and hospitalization is required for those who have caught the disease and those who have recovered should still receive a full course of the diphtheria vaccine to prevent a recurrence because contracting the disease doesn't guarantee lifetime immunity.

CORONARY/ ISCHAEMIC HEART DISEASE

This chapter brings a brief background of the Ischemic Heart Disease covering the history and epidemiology of the disease, its presentation, methods of treatment or control and the public health implications of the disease.

Introduction

Common Name: Coronary Artery Disease, Medical Term: Ischaemic Heart Disease. Ischemic or ischemic heart disease (IHD), or myocardial ischemia, is a disease characterised by reduced blood supply to the heart muscle, usually due to coronary artery disease (atherosclerosis of the coronary arteries). Its risk increases with age,

smoking, hypercholesterolemia (high cholesterol levels), diabetes, hypertension (high blood pressure) and is more common in men and those who have close relatives with Ischemic heart disease.

Symptoms of stable Ischemic heart disease include angina (characteristic chest pain on exertion) and decreased exercise tolerance. Unstable IHD presents itself as chest pain or other symptoms at rest, or rapidly worsening angina. Diagnosis of IHD is with an electrocardiogram, blood tests (cardiac markers), cardiac stress testing or a coronary angiogram. Depending on the symptoms and risk, treatment may be with medication, percutaneous coronary intervention (angioplasty) or coronary artery bypass surgery (CABG).

It is the most common cause of death in most Western countries, and a major cause of hospital admissions. There is limited evidence for population screening, but prevention (with a healthy diet and sometimes medication for diabetes, cholesterol and high blood pressure) is used both to prevent IHD and to decrease the risk of complications.

Description

Coronary artery disease is a condition in which fatty deposits (atheroma) accumulate in the cells lining the wall of the coronary arteries. These fatty deposits build up gradually and irregularly in the large branches of the two main coronary arteries which encircle the heart and are the main source of its blood supply. This process is called atherosclerosis which leads to narrowing or hardening of the blood vessels supplying blood to the heart muscle (the coronary arteries). This results in ischaemia(inability to provide adequate oxygen) to heart muscle and this can cause damage to the heart muscle . Complete occlusion of the blood vessel leads to a heart attack (myocardial infarction).

In the United States, cardiovascular disease is the leading cause of death among both sexes, and coronary artery disease is the commonest cause of cardiovascular disease.

Myocardial infarction causes 35% of deaths in men between 35 and 50.The death rate is higher for men than for women between the ages of 35 and 55.However, after the age 55, the death rate for men declines but the rate for women continues to climb.

Causes

Exact cause is unknown. However there are a number of risk factors. Control of these risk factors has been shown to reduce the severity and complications of the disease.

Prevention

It is now clear that reducing certain risk factors, we can both prevent coronary artery disease and delay its progression and complications after it it has become manifest. Treatment of lipid abnormalities has now been shown to delay the progression of atherosclerosis and in some cases has even produced regression of the atherosclerotic plaques.

Signs and symptoms
Early stages: No symptoms. Later stages: Angina pectoris (burning, squeezing, heaviness, or tightness in the chest that may extend to the left arm, neck, jaw, or shoulder blade). See Angina Pectoris. Typically, angina is precipitated by physical activity, lasting no more than a few minutes, and is relieved by rest. Usually angina is worse when exertion follows a meal. It is also worse in cold weather and can be triggered by walking from a warm room into the cold air. Emotional stress can also cause or worsen angina. Not all people with ischemia will present with angina. Ischemia without angina is called silent ischemia. It is not yet understood why ischemia is sometimes silent.

Risk Factors
The aetiology of IHD is multi-factorial. It is the result of interaction between genetic, lifestyle and environmental factors.

Age
IHD increases with age. This is a non-modifiable risk factor.

Gender
Traditionally, IHD has been considered a disease of men. However, IHD is the leading cause of death in both men and women. It is responsible for a third of all deaths in women worldwide and half of all deaths in women over the age of 50 in developing countries.

Social deprivation
In England and Wales there is a positive correlation between deaths from circulatory diseases and levels of deprivation. There is a marked difference in prevalence of IHD between and within communities. Men and women living in the West of Scotland are nearly six times more likely to die prematurely from coronary heart disease than men and women living in the South West of England. Within London, people living in Tower Hamlets have a three times increased risk of dying prematurely from IHD than those in Kensington and Chelsea. The difference in IHD rates in different socio-economic groups is related to many factors including diet, smoking, exercise, alcohol.

Smoking
Mortality from IHD is 60% higher in smokers. Regular exposure to passive smoking increases IHD risk by 25%. About 1 in 8 deaths from cardiovascular disease (CVD) were attributable to smoking in 2000 in the UK. WHO research estimates that over 20% of CVD is due to smoking.

Poor nutrition
A World Health Organisation report in 2003 stated that a diet high in fat (particularly saturated fat), sodium and sugar and low in complex carbohydrates, fruit and vegetables increases the risk of cardiovascular disease. COMA recommended in 1994 that the percentage food energy derived from fat should be 35%, with 11% from saturated fat. The Scientific Advisory Committee on Nutrition suggests that salt intake should be no more than 6g per day. The National Diet and Nutrition Survey in 2000/01 found that the total energy intake from fat was 36% in men and 35% in women with 13% from saturated fat. It also found that the average intake of fruit and vegetables was less than 3 portions per day compared to the recommended 5 portions. In the same survey salt intake was 11g per day for men and 8.1g for women. There are national, regional, socio-economic and ethnic differences in nutrition. Tran's fatty acids reduce HDL and increase LDL-cholesterol and can increase coronary heart disease risk. A meta-analysis showed that a 2% increase in the energy intake from trans-fatty acids increased IHD incidence by 23%. Eating oily fish rich in omega-3 fatty acids has been shown to reduce IHD mortality. Increased intake in dietary fibre also appears to reduce risk.

Physical activity infrequent exercise
Physical activity reduces the risk of IHD. The 2002 World Health Report estimated that over 20% of IHD in developed countries was due to physical inactivity. Recommended physical activity levels are 30 minutes of moderate physical activity on 5 or more days per week. In 2003, over one third of UK adults were inactive (exercised for less than one occasion of 30 minutes per week).

Alcohol
1 to 2 units of alcohol per day reduces the risk of IHD. Alcohol increases HDL cholesterol and reduces thrombotic risk. Higher levels of consumption increase risks from other causes. The World Health Report in 2002 estimated that 2% of IHD in men in developed countries is due to excessive alcohol consumption. Men should drink no more than 3 to 4 units on any one day and women no more than 2 to 3 units.

Psychosocial well being
Work stress, lack of social support, depression, anxiety and personality (particularly hostility) can all increase IHD risk.

Blood pressure
For adults aged 40 to 69 years, each 20 mmHg rise in usual systolic blood pressure or 10 mmHg rise in diastolic blood pressure doubles the risk of death from IHD. The INTERHEART study showed that 22% of heart attacks in Western Europe were due to a history of high blood pressure and those with hypertension had almost twice the risk of a heart attack.

Cholesterol
IHD risk is related to cholesterol levels. The INTERHEART study suggested that 45% of heart attacks in Western Europe are due to abnormal blood lipids. People with low levels of HDL-cholesterol have an increased risk of IHD and a worse prognosis after a myocardial infarction. In the UK, it is suggested that the target cholesterol is < 4 mol/l for people with diabetes or established CVD or for people at high risk of developing CVD. People with HDL-cholesterol < 1 mol/l should also be considered for treatment.

Overweight and obesity
Obesity is an independent risk factor for IHD. It is also a risk factor for hypertension, hyper-lipidaemia, diabetes and impaired glucose tolerance. Central or abdominal obesity is most significant. Those with central obesity have over twice the risk of heart attack.

Diabetes
Men with Type 2 diabetes have a 2 to 4 times greater annual risk of IHD; women have a 3 to 5 time greater risk. Over 4% of men and 3% of women in England have diagnosed diabetes. The prevalence is increasing.

Ethnicity
South Asians living in the UK (people from India, Pakistan, Bangladesh and Sri Lanka) have a higher premature death rate from IHD (46% higher for men; 51% higher for women). Hypotheses for this include migration, disadvantaged socioeconomic status, 'proatherogenic diet', lack of exercise, high levels of homocysteine and LP(a) lipoprotein, endothelial dysfunction and enhanced plaque and systemic inflammation. The premature death rate from IHD in West Africans and people from the Caribbean is much lower (half the rate compared to the general population for men and two-thirds of the rate for women).

Family history
First degree relatives of patients with premature myocardial infarction have doubled the risk themselves. Premature coronary heart disease is that before 55 years in men and 60 years in women. More than one third of admissions for premature myocardial infarction could be prevented by screening and treating first degree relatives. Genetic predisposition and shared lifestyle are likely to contribute. Several regions of the human genome have been shown to be associated with either IHD or hypertension.

Serum homocysteine
It was previously thought that elevated levels of homocysteine is an independent risk factor for IHD, likely to due oxidative damage to endothelium, platelet activation and thrombus formation. The theory was that dietary supplementation with folic acid could reduce homocysteine levels and therefore IHD incidence. A meta-analysis in 2005 disputed this.

Diagnosis and treatment
Diagnosis of angina is a clinical diagnosis based on a characteristic complaint of chest discomfort or chest pain brought on by exertion and relieved by rest. Confirmation may be obtained by observing reversible ischemic changes on ECG during an attack or by giving a test dose of sublingual nitro-glycerine that characteristically relieves the pain in 1 to 3 minutes.
Certain tests may help determine the severity of ischemia and the presence and extent of the coronary artery disease. Diagnostic tests may include electrocardiogram (measures electrical activity of the heart), echocardiogram (measures sound waves), exercise-tolerance test, thallium stress test, blood studies to measure total fat, cholesterol and lipoproteins, X-rays of the chest and coronary angiogram (cardiac catheterization).

General measures

-Stop smoking
-Treat elevated cholesterol levels with low fat, low cholesterol diet, exercise and cholesterol lowering medications
-Treat elevated blood pressure
-Reduce stress
-Maintain ideal body weight
-Balloon angioplasty (treatment for obstructed arteries, especially those supplying blood to the heart and brain. A small uninflated balloon is passed up the artery to the obstruction, and then expanded to release the obstruction
Although these procedures may decrease or eliminate symptoms for a while, they do not control the underlying disease. Surgery to bypass coronary arteries (severe cases).
End-stage coronary artery disease, even when no simple procedures will help, can still be cured with a heart transplant in rare cases.

Medications
Four types of medications are available: beta-blockers, nitrates, calcium channel antagonists and anti-platelet drugs.

Beta-blockers reduce the resting heart rate and so reduce the demand for oxygen. Beta-blockers and nitrates have been proven to reduce the incidence of heart attacks and sudden deaths in people with coronary artery disease.

Nitrates -such as nitro-glycerine, cause dilatation of the blood vessels. There are short-acting and long-acting nitrates. Nitro-glycerine is available as a tablet (sublingual) or an oral spray. A tablet of nitro-glycerine placed under the tongue or inhalation of the oral spray usually relieves an episode of angina in 1 to 3 minutes-the effect of these short-acting nitrates lasts 30 minutes. Anyone with chronic stable angina must keep nitro-glycerine tablets or spray with them at all times.

Long-acting nitrates are available as tablets, skin patches or paste. Tablets are taken 1 to 4 times daily. Nitro paste and skin patches, in which the drug is absorbed through the skin over many hours, are also effective. Long-acting nitrates do tend to lose their effectiveness when taken regularly and therefore it is recommended to have 8 to 12 hour interval without taking the drug to maintain its effectiveness.

Calcium channel antagonists prevent the blood vessels from constricting and thus prevent coronary artery spasm. Certain calcium antagonists, such as vera-pamil and diltiazem, also slow the heart rate and in some patients these drugs are used in conjunction with beta-blockers to prevent episodes of tachycardia (fast heart rate).

Anti-platelet drugs such as aspirin are recommended for patients with coronary artery disease. Aspirin binds irreversibly to platelets and prevents them from clumping on blood vessel walls-thus preventing platelets from forming a clot on the fatty plaques which could block an artery and result in heart attack. Recommended dose is one baby aspirin or half an adult aspirin daily. For people with allergy to aspirin can be treated with .alternative medications such as ticlopidine or clopidogrel bisulphate

Activity
Engage in a program of moderate, daily physical exercise. Resume sexual activity once medical permission is given.

Diet
Low-fat and low cholesterol diet. If you are overweight, begin a moderate reducing diet and stick to it.

Possible complications
Angina pectoris
Life-threatening myocardial infarction (death of heart muscle cells from inadequate blood supply).
Sudden death

Prognosis

Treatment can prolong life and improve its quality. Tremendous amount of research in this field, and new advances are being made and increasing evidence that aggressive treatment can reverse or arrest course of this disease. It is very important to follow your doctor's instructions, especially with respect to lifestyle changes and cholesterol reduction.

Long term prognosis depends on a number of key factors such as the age, the extent of coronary artery disease, the severity of symptoms and most of all , the pumping ability of the heart.

Epidemiology of IHD
Incidence and Prevalence

Mortality rates

Coronary heart disease (IHD) is the most common cause of death (and premature death) in the UK.
1 in 5 men and 1 in 6 women die from IHD.
There are 101,000 deaths from IHD in the UK each year.
Death rates from IHD have fallen by 46% for people under 65 years in the last 10 years. This fall is fastest in those aged 55 and over. It is largely due to a reduction in major risk factors (mostly smoking) and improvement in treatment and secondary prevention. The fall is not as high as that in some other countries such as Australia (48%) and Norway (54%).
Death from IHD is more likely during winter.

Morbidity rates

The average incidence of myocardial infarction is 600 per 100,000 in men aged 3069 and 200 per 100,000 in women. The incidence increases with age.
There are about 52,000 new cases of angina per year in all men living in the UK and about 43,000 new cases in women.
About 4% of men and 2% of women in the UK have had a heart attack.
Prevalence increases with age and is higher in men.
About 8% of men and 5% of women aged 55 to 64 and 17% of men and 8% of women aged 65 to 74 have or have had angina.
The prevalence of IHD is about 7.4% in men and 4.5% in women.
The prevalence is higher in Scotland (4.6%) than in Wales (4.3%) or England (3.6%).
The prevalence is higher in the North of England and Wales than in the South of England.
The prevalence is higher in lower socio-economic groups.
Of note, mortality from IHD is falling but morbidity appears to be rising.

Economic cost

IHD is estimated to cost the UK economy over Â£7.9 billion a year (including direct health care costs and productivity losses).
Among the more developed countries in Europe, only Ireland and Finland have a higher rate of IHD than the UK.

INJURIES

Introduction
Injuries are a long standing public challenge and require intervention to deal with them. In the following resources, we will present the case of injuries.

What problems and challenges are caused by injuries?
What are the known or acceptable methods of control (available programs of main intervention)?
What are the emerging issues around this condition?

Definition of injuries
Injury is a damage or harm caused to the structure or function of the body due to an external agent or force; the agent may be physical or chemical. Injuries may be accidental or intentional. Intentional injuries are usually violence related.

Globally, about 16 000 people die of injuries every day and about 5.8 million people die every year. This corresponds to an annual mortality rate of 97.9 per 100 000 population. World Health Report 2002, injury accounts for 12.2% of total burden of disease. Road traffic injuries account for 25% of all deaths from injury. In comparison to other diseases and health conditions, injury morbidity, mortality and disability account for disproportionate deaths among children and young adults. This leads to a major burden on health sector and social welfare services, and its economic consequences for the care as well as loss of productivity.

Types of injuries
Injuries fall into six types; burns and scalds (that are due to flames, steam, radiations and hot liquids), electric shock, drowning and near drowning, falls, road traffic accidents, air travel injuries, bites, diving injuries, poisoning and occupational (work related). The most vulnerable groups for road traffic injuries are pedestrians, pedal cyclists and motorcyclists.

Grading of injuries
Injuries are in two main grades, minor and severe. Most injuries are minor and are not life threatening. They can be managed using first aid methods and hospitalization is rarely required. *Minor injuries* include: - bruises, superficial wounds, superficial burns, concussion, strain and sprain.

- Bruise is a haemorrhage that is under the skin caused by contusion.
- Wound is as a result of cuts to and grazes under the skin that can cause bleeding and lacerations.
- Burns are injuries that are caused by excess heat, chemical exposure, or sometimes cold (frostbite)

- Concussion is the type of injury that gives mild traumatic brain injury caused by a blow usually without any penetration to the skull or brain
- Sprain is the type of injury that occurs to ligaments caused by the sudden over stretching.
- Strain on the other hand is the type of injury that occurs to muscles caused by the sudden over stretching.

Severe injuries are: head injuries apart from concussion, fractures, deep wounds, traumatic amputation and any injury that involves an organ. Signs of severe injuries include severe pain; swelling; fever; numbness; deep wounds; wounds over bones; inability to use the injured area.

- Fractures are injuries that occur to the bones.
- Shock is a serious medical condition that arises from tissues not getting sufficient oxygen and nutrients.
- Amputation is the removal of part arising from trauma or surgery.
- Joint Dislocation is the displacement of a bone from its normal joint position.

Classification of Injuries
Injuries are classified basing on the WHO scheme (ICECI) that is a multi-axial and hierarchical structure. It is based on seven modules:

- Mechanisms of injuries
- Objects/substances causing injuries
- Place of occurrence
- Activity when injuries
- The role of human intent
- Use of alcohol
- Use of other psycho-active drugs.
- Occupational injury).

However, in addition to the above modules there are modules that are used collect additional data on special topics (violence, transport, place, sports and occupational injury).

Clinical presentation of injuries
This usually depends on the type and severity of the injury. In minor injuries, there pain, swelling, heat, reddening, lack of function and major injuries, there is shock, severe pain, heavy bleeding, obvious loss of function which is sometimes permanent like blindness and sometimes loss of consciousness.

Management of injuries
The management of injuries like clinical presentation also depends on the type and severity of the injury. For example minor injuries can be managed using simple measures such as first aid and out-of-hospital or as out patients. The

management is usually simple and less costly as compared to the management of major injuries.

Major injuries usually require hospitalization and expensive in terms of resource use and management. The patients need a lot of time for injury management before recovery/ healing and they normally end up being impaired or disabled or handicapped or all the three.

Impacts of injuries

-Impacts of injuries can be grouped into; physical, social, economic and psychological.

-*Physical effects* include loss of body parts and loss of/reduction of function and death.

-*Social effects* include stigma, separation from family and friends, in some cases traditional divorce, marriage break-ups, inability to participate in leisure activities and loss of effective participation on social events. Death brings about bereavement.

-*Economic effects* is loss of job, loss of time while in hospital, loss of productivity or reduction in productivity, loss of income, increased expenditure on medical care and hospitalisation for injured. Reduction of income may bring about transition towards poverty, especially in middle and low income groups. Injuries economically, by loss of government revenue from taxation of income of citizens who have jobs and income. Increased bed occupancy leads to congestion in hospitals and subsequent increase of nosocomial infections. This increases work loads on health workers leading decline of quality of care.

-*Psychological effects* cause depression, anxiety, fear and Post-Traumatic Stress Disorder (PTSD).

What are the known or acceptable methods of controlling injuries?
There are five domains that should be talked and looked into in order to control injuries in any community or society.

1. Improvement in local environment
2. Legislation
3. Public education
4. Product safety
5. Improvement of the levels and quality of emergency care.

Prevention of traffic road accidents and injuries
Accidental injuries also known as intentional injuries can be prevented by designing a safe and sustainable road traffic system; reducing motor vehicle

traffic; encouraging use of safer modes of travel; safety awareness in planning road networks; incorporating safety features into road design; remedial action at high risk crash sites; improving the visibility of vehicles; using crash protective vehicle design; setting and securing compliance with key road safety rules; setting and enforcing speed limits; installing cameras at traffic lights; regulating drivers hours of work in commercial and public transport systems; setting and enforcing mandatory crash helmet use; seat belts and child restraints; control of drink-driving.

Household preventive measures
This are some of the measures that should be put in place; keeping all chemicals out-of-reach of children, use safe stoves for cooking, putting in place fire safety measures such as raising fire places and other physical barriers that do not allow children to access certain household items, use of ramps for persons who are physically challenged, building and use of fire escapes and putting in place fire-assembly areas in case of fires and fire drills. There should be deliberate effort on the side government to legislate and enforce building and engineering regulations and engineering professional practice.

Preventive measures of injuries in recreation places
Design of recreation centres and facilities that have safety measures taken care-of. Swimming pools must have clear instructions on their usage.

Prevention of violence related injuries
Violence related injury is also known as intentional injury. The ways of preventing are; promotion multi-media campaigns to promote non-violence social norms, restricting access to fire arms, enforcement of liquor and licensing laws, reduction of available alcohol through and pricing, pre-school enrichment programs to give children an educational head start. Life skills trainings like in swimming, communicating emergencies and resuscitation. Conducting parents training on child development, non-violent disciplining and problem-solving skills. Professional nurses and social workers should carry out home visiting to inspect the safety of the homes.

The public health approach to injury prevention has various major aspects that include:

- Defining the problem before we can address an injury problem, we need to know how big the problem is, where it is, and whom it affects. We also need to know why and what factors put people at risk for that injury? And conversely, what factors protect people from it?
- Identify risk and protective factors
- Develop and test prevention strategies In this step, we put knowledge into action. Using the information gathered in the previous steps develop strategies to prevent particular injury problems. We implement these

strategies in communities that are experiencing the problem. And we study the effects of these strategies to determine whether and how well they are working. We use this information to identify any elements we need to change to eliminate difficulties or increase effectiveness.

- Intersectoral coordination should be setup for overseeing/developing injury prevention and control strategy.. Injury prevention programme should be integrated within the policies of various organizations such as schools, hospitals, factories etc.
- Referral system: hospitals should separate trauma centre in each zone with trained staff and adequately equipped facilities with definite guidelines/flow chart for injury management and referral. For surveillance of injury, trauma registry should be initiated at these centres.
- Training: Training should be imparted in relation to emergency management of injury at under graduate/post-graduate levels of medical schools as well as periodic orientation practical training to dispensary doctors/private practitioners. Capacity of nurses and paramedics need to be strengthened as well. Various organizations and schools should have fire-fighting measures and under go periodic drills of fire fighting.
- Health education: Public health education awareness campaigns should be initiated regarding the hazards of injury and its prevention. Health education should be included in school curricula and awareness spread through mass media, social workers and health personnel. For this mass education campaign, there should be a separate unit for IEC (information, education, and communication) activities. Modification of environment and infrastructure safety are the longer term investment for sustainable safety.
- Assure widespread adoption What we learn in the developing and testing step has little benefit if we keep the information to ourselves. In this final step of the public health approach, share the knowledge with the public through sensitization and provide funding or expert consultation so that communities can replicate these successful strategies
- Obtaining political commitment which is vital for enforcement of regulations and law.

Emerging issues

- Substance abuse and misuse for intoxication
- Use of corrosive liquids like acids to burn people
- Toxic effects of new drugs
- Terror attacks like arson in schools and bombs in buses
- Dangerous effects of medicine
- Inadequate staffing
- Inadequate training and inadequate equipment to handle injuries
- Resistance of wound infections to available drugs and antibiotics

- Poor supervision of building works and poor control of construction materials causing construction related injuries and death (Bwebajja, Nalya SS, and NSSF Pensions Towers)
- Issues concerning manufacture of poor standard household items and foods
- Displacement of populations
- Increasing levels of poverty
- Domestic violence

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