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CHRISTIAN RELIEF & DEVELOPMENT ASSOCIATION

TRAINING PROGRAMME
ON
THE MANAGEMENT OF ACUTEFEBRILE ILLNESS

CCRDA

Prepared in collaboration with
Ministry of Health, & The Facutly of
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INTRODUCTION TO FEVER

"Fever is only a symptom and we are not sure it is an enemy. It may be a friend." (Du Bois)

WHAT IS FEVER?

The body temperature is normally maintained around 37°C (range 35.9 - 37.2°C), and controlled by the hypothalamus, which is a part of the brain. The body temperature may vary slightly between individuals and at different times of the day and night. The body temperature may rise due to exercise, digestion, sudden increase in environmental temperature, excitement and after ovulation. When it is very hot outside, the body looses heat by sweating in order to maintain body temperature. In cold weather the body shivers.

Sometimes the body is unable to maintain a normal temperature. The result will be either **hypothermia** (lower than normal body temperature) or **hyperthermia** (a higher body temperature), which is sometimes experienced by people working in very hot climates. **Fever** (also called **pyrexia**) is a rise of body temperature above normal as a result of disease. It is not dependent on environmental temperature, but it is a response to disease, when certain fever producing substances are formed which act on the hypothalamus. This may benefit the body. There is increasing evidence that the body's defence against infection work better at above normal temperatures. However prolonged fever over 41°C may result in permanent brain damage and markedly elevated or prolonged fevers may result in metabolic disturbances..

HOW DO WE RECOGNISE FEVER?

We can feel whether someone is feverish by putting our hand on the forehead and compare the persons skin temperature with the one of our hand. More accurately we measure body temperature with a **thermometer**. This can be done: - in the rectum (rectally).
- under the armpit (axillary).
- under the tongue (orally).

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The rectal temperature is the best measure of the body temperature. After shaking the thermometer down and lubricating it with vaseline, the thermometer is carefully inserted for a distance of 3 cm into the rectum in an infant and 5 cm for older children and adults, and read after 3 minutes. After reading the temperature, wash the thermometer thoroughly and allow it to dry before re-using.

The oral temperature is influenced by solutions recently taken by mouth. A full minute under the tongue should be the minimum time allowed for the procedure. This method is not advised below the age of 6 years.

The axillary temperature is about 0.5°C lower than orally and 1°C lower than rectally. It is useful in newborn infants and young children.

DEFINITION OF FEVER BY TEMPERATURE		
Classification	**Rectal Temperature**	**Axillary Temperature**
Low body temperature (hypothermia)	less than 35.5°C	less than 35°C
Normal temperature	35.5-37.7°C	35-37.2°C
Low fever	37.7-38.9°C	37.2-38.4°C
High fever	39°C or more	38.5°C or more

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ACUTE AND CHRONIC FEVERS

When a patient presents with a complaint of fever, it can be difficult to find the cause of the fever. It is helpful to make a differentiation in acute and chronic fevers. Fevers of more than 2 weeks duration we call chronic or prolonged fevers and those lasting less than 2 weeks acute fevers. Sometimes chronic fevers have a relapsing pattern, in which a period of fever is followed by an afebrile period.

The characteristics of the temperature pattern (graphic record) may be of diagnostic value, especially when viewed in the light of other clinical findings, and may serve as a guide to the effectiveness of treatment.

Pyrexia of Undetermined Origin (PUO) is a difficult problem in developing countries because of lack of sophisticated techniques for investigation. A detailed history and good physical examination may give you important clues to the diagnosis. Localising signs and symptoms can indicate the site of an infection, such as sore throat, cough, bloody diarrhoea, joint or bone pain, dysuria and loin pain. The commonest cause of PUO in the tropics is cryptogenic tuberculosis (disseminated tb without evidence of its origin).

CLASSIFICATION OF CAUSES OF FEVER

1. Infections (e.g. bacterial, viral, parasitic, rickettsial).
2. Collagen diseases (e.g. rheumatic fever, rheumatoid arthritis).
3. Malignant neoplastic diseases (e.g. any malignant tumour).
4. Central nervous system diseases (e.g. stroke, head injury).
5. Haematologic diseases (e.g. lymphoma, leukaemia, haemolytic anaemias).
6. Cardio-vascular diseases (e.g. myocardial infarction, thrombosis).
7. Gastro-intestinal diseases (e.g. liver abscess).
8. Endocrine diseases (e.g. hyperthyroidism).
9. Diseases due to physical agents (e.g. heat stroke).
10. Diseases due to chemical agents (e.g. drug reactions, allergic reactions).
11. Disorders of fluid balance (e.g. dehydration).
12. Other diseases (e.g. sarcoidosis).
13. Unknown causes.

WHEN TO TREAT FEVER?

Fever is a symptom of an underlying disease, so it is important to treat this disease if necessary. Symptomatic treatment for low and moderate fever is not necessary. It is the underlying disease, not the fever, that causes ill health. However temperatures in excess of 41°C are a medical emergency.
Reduction of fever is important in the following circumstances:

- High fever of 39°C or more.
- Fever in case of severe pneumonia that requires oxygen therapy (lowering the body temperature decreases the body's need for oxygen).
- When the patient is very uncomfortable due to the fever.

HOW TO TREAT FEVER?

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The principal problem is to determine and treat the underlying cause of the fever. Besides this there is symptomatic treatment of fever.

Make sure the patient is in a cool (but not cold) place where there is plenty of air circulating.

Patients with fever need to drink extra fluids to compensate for fluid and electrolyte loss from increased perspiration.

Drugs which bring down fever are called **antipyretics**. Acetyl salicylic acid (aspirin) and paracetamol are the ones best known. The drug of choice to reduce fever in young children is paracetamol, but don't give paracetamol to infants under two months of age.

Cooling a patient with tepid sponging is also effective to bring down high fever. It is to be avoided when the patient feels and looks cold.

Alcohol should never be rubbed on the skin to cool a child with fever. If alcohol is absorbed through the skin it can be very toxic in a young child.

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A DIAGNOSTIC APPROACH TO ACUTE FEBRILE ILLNESSES

IS THE FEVER ACUTE OR CHRONIC?

When presented with a patient with fever, it is useful first to determine whether the fever is acute or prolonged. Fevers of more than 2 weeks duration, we call chronic or prolonged fevers. Sometimes the same organism may cause fever for a week, sometimes for a few weeks, such as typhoid fever which in some individuals causes a mild fever of only a few days, in others a continuous fever for three weeks.

Helpful simple laboratory investigations in fever are:

- Thick blood film for malaria parasites.

- Total (and differential) White Blood cell Count (WBC).

IS THE FEVER CAUSED BY MALARIA?

In holo-endemic malarial areas, positive blood films are so common and people may have parasites in their blood without having fever, that it is diffiult to determine whether the fever is caused by malaria. For practical reasons, treat the malaria when the blood film shows malaria parasites and see what happens.

FEVER WITH A NEGATIVE MALARIA BLOOD FILM

The results of the WBC divides this group into two categories:

1. With Polymorpho Nuclear Leucocytosis (PNL).
   This group includes:
   - Pyogenic infections.
   - Leptospiral infection.
   - Relapsing fevers.
   - Amoebic liver abcess.
   - Acute collagen diseases.

2. Without PNL.
   This group includes:
   - Viral infections.
   - Rickettsial infections.
   - Typhoid.

It is important to look for organ specific localising signs and symptoms, which can give a clue to the diagnosis.

1.a: Acute fever with PNL and localising symptoms

- Painful throat:
  - streptococcal tonsillitis
  - diphteria

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- Cough, chest pain, sputum: ° pneumonia
- Pain and swelling of a joint: ° septic arthritis
- Frequency, dysuria, loin pain: ° pyelonephritis
- Headache, neck pain, neck stiffness: ° meningitis
- Severe bone pain: ° osteomyelitis
- Severe lower abdominal pain: ° pelvic inflammatory disease
- Bloody diarrhoea: ° bacillary dysentery
  ° campylobacter infection
- Localised enlarged lymph gland ° local sepsis
  ° plaque
- Well defined inflammation of skin: ° erysipelas
- Ill defined subcutaneous inflammation: ° cellulitis

1.b: Acute fever with PNL and no localising symptoms

- Septicaemias of all kind.
- Leptospirosis.
- Relapsing fever (tick-borne or louse-born).
- Acute non-typhoid Salmonella septicaemia.

2.a: Acute fever without PNL and without malaria parasites

- Viral infections (e.g. hepatitis, dengue).
- Rickettsial infections (typhus).
- Typhoid fever.

Other important non-localising symptoms accompanying fever:

- Rash.
- Generalised lymph gland enlargement (lymphadenopathy).
- Enlarged spleen (splenomegaly).
- Anaemia.

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- Jaundice.

- Poly-arthritis.

CASE STUDY 1:

On saturday morning Sister Bezunesh was sitting on the verandah of her house when she was called to the clinic to see an Italian lady who was on tour through Ethiopia and had developed a fever.

She had spent some days in Addis Ababa and visited her sister in Arba Minch and together they had travelled to Awassa and Langano. On her way back she had high fevers for three days, loss of appetite, headache, and watery diarrhoea. She had not had any vaccinations before she left Europe and had no medicines with her. She was five months pregnant.

On examination Sister recorded a temperature of 39°C, with a BP of 80/55, and a pulse of 110. The woman looked unwell, was moderately dehydrated and vomited. Her fundus was 24 weeks and the foetal heart was heard. There were no other abnormal findings.

1. What is the most likely diagnosis?

2. What treatment would you give?

The woman was diagnosed as having malaria and admitted in the clinic, and was treated with oral chloroquine, of which she vomited the first dose immediately, so that a chloroquine injection was given. Apart from this she was given aspirin, ORS, and soup.

3. What is the dosage of a chloroquine injection
   - for adults?
   - for children?

4. When is a chloroquine injection indicated?

In the late afternoon Sr. Bezunesh returned to the clinic to check her patient. The woman had had three episodes of watery diarrhoea with blood, and had collapsed in the bathroom. Her temperature was now 40°C, BP 70/40, P 130, and there were irregular uterine contractions.

5. What is your diagnosis now?

6. How would you manage this case?

Sr. Bezunesh put the patient in bed and put up an IV drip with Ringers lactate and elevated the foot end of the bed. She checked a stool specimen under the microscope and didn't find any amoeba, but saw many rbc's and wbc's.

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7. What is the likely cause of the bloody diarrhoea?

8. What would she have seen under the microscope when the diarrhoea was due to amoebic dysentery?

9. What treatment would you give?

Sister gave her amoxycilline and continued chloroquine. The next morning her general condition had improved, though she was still having slight irregular contractions. the foetal heart was still heard. The drip was discontinued and other treatment continued. Three days later she was discharged from the clinic.

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CASE STUDY 2

Dr. Tesfaye was called to see a 30 years farmer who had been referred to the hospital from a rural clinic because of a four days history of fever with severe pain over the liver. There was no nausea or vomiting. There was a history of several episodes of bloody diarrhoea during the last few months.

The temperature was 39°C and the liver was enlarged to 2 cm below the costal margin and extremely tender. he was not jaundiced.

1. What diagnosis do you think most likely?

2. What investigations would help you to make a diagnosis?

The WBC was 6,400, with a relative monocytosis, ESR: 10 mm. The patient became deeply jaundiced two days later and the fever, severe pain and liver tenderness gradually subsided. His liver function tests were typical of viral hepatitis. His serum was later positive for hepatitis B surface antibody.

3. What is the treatment?

4. What advice would you give to the patient?

5. What late complications do you know of hepatitis B infection?

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CASE STUDY 3

Dr. Tesfaye was feeling happy and was whistling when he walked back from his lunch to the hospital for his afternoon outpatient clinic. Soon he would start his annual leave and he had fixed up to play golf with some friend as soon as the clinic would be over. However the clinic was extremely busy and his mood progressively darkened. By the time the last patient was shown, it was already 5.30 pm.

She was a 34 years old lady from a rural village, referred to him by the health assistant. She was complaining of cough, diarrhoea, and fever of uncertain duration. Dr. Tesfaye could see from his across-the-desk initial "examination" that she was thin and unwell, so he promptly admitted her "for investigation" and dashed off to the golf club.

1. What initial investigations would you consider in such a case?

The next day Dr. Tesfaye was in a worse mood. He had played very badly the previous evening. Also the ward doctor - Dr. Fekade - was absent that day so he had to decide on some tests for his new patient himself. Sr. Tsehai brought him the results. The chest Xray showed bilateral mid- and lower zone shadowing; the first of three sputa was negative by gram and Ziehl-Neelsen staining; a slide for malaria was negative; stool microscopy had shown cysts of Entamoeba histolytica and larvae of Strongyloides stercoralis. In addition the Hb was 10,5 gr%; WBC: 2,1x 10^9/l; ESR: 65 mm.

Dr. Tesfaye was very pleased with the results and ordered penicillin, metronidazole, and thiabendazole. As an afterthought he added iron injections and vitamin tablets.

2. What has he still not done?

3. What do you think of his treatment?

Sister Tsehai was not entirely convinced that things were going quite as they should and was therefore pleased when Dr. Fekade came back on the ward later in the day. After making his usual cup of coffee, she tactfully suggested he might be interested to "review" Dr. Tesfaye's patient.

Dr. Fekade noticed how unwell the patient looked, and found out that the illness had been quite long standing and progressive. On examination, she had obviously lost a great deal of weight, she was febrile (38°C), and was breathing rapidly with widespread crepitations in both lungs. He also noticed general enlargement of lymph nodes and on her left flank there was a large area of rather thickened skin with three or four well-defined, firm nodules.

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Dr. Fekade presented his findings to Dr. Tesfaye on the next day's ward round. Dr. Tesfaye did not seem impressed, but said that one of the lymph nodes and one of the nodules should be biopsied. Dr. Fekade, however, suggested the diagnosis he thought most likely, at which Dr. Tesfaye hastily withdrew his biopsy suggestions and agreed to a single blood test and changed the treatment.

4. What was the diagnosis and what was the test?

5. What was the nodular eruption?

6. What treatment change did Dr. Tesfaye make?

7. What are the major and minor symptoms of this disease and how can you make a clinical diagnosis without test facilities?

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CASE STUDY 4

A 15 years old boy was brought to the rural clinic of Sr. Bezunesh because of 6 days fever, general malaise, headache, and abdominal pains. The parents had already given some chloroquine which they bought with a local rural drug vendor, but there was no improvement. He had no diarrhoea, even hadn't opened his bowels for 3 days.

On examination the boy looked quite sick, a bit drowsy and out of touch. a temperature of 39.8°C was recorded; BP 110/70; P 72. He didn't look anaemic or jaundiced. The spleen was three fingers palpable below the costal margin, and the abdomen was generally tender but soft and not distended. Otherwise no abnormalities were observed.

Sister admitted the boy under the diagnosis "chloroquine resistant malaria" and prescribed quinine 600 mg 8 hourly, aspirin, and tepid sponging.

1. What do you think of this diagnosis?

2. What additional questions would you ask to verify this diagnosis?

Despite this treatment after three days the fever remained constantly between 38.5 - 40°C and he developed a slight cough and complained of deafness and ringing in the ears. He looked rather toxic, was neither neckstiff nor short of breath, but on auscultation of the chest Sister thought she heard slight wheezing, and prescribed ampicillin and ephedrin tablets for the bronchitis.

3. Do you agree with the diagnosis bronchitis?

4. What diagnosis do you consider?

5. What do you think of his deafness?

The next day the boy's condition had seriously deteriorated. He had vomited several times and complained of severe abdominal pain. The boy looked weak and moderately dehydrated with sunken eyes, and his abdomen was extremely tender with guarding all over. Sister rushed to Abba Gebre Mariam to borrow his old landrover, and the boy was taken to the regional hospital, where he was admitted in the surgical ward, given an IV drip and prepared for emergency laparotomy because of peritonitis.

6. What is the likely cause of the peritonitis?

7. What medication would you give to the boy to treat his condition?

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8. What complications do you know of his disease?

CASE STUDY 5

A 5 months old baby was brought to the hospital one evening by his mother with a history of fever for three days, continuous cough, and of not feeding. dr. Tesfaye recorded a temperature of 38°C, marked shortness of breath with a respiration rate of 50/min, slight chest indrawing, and fine crackles (creps) all over the chest, and admitted the child.

1. What is your initial diagnosis?

2. What treatment would you prescribe?

The child was put on i.m. penicillin. After two days the child was no better, and had a generalised convulsion in the afternoon. Dr. Tesfaye ordered paraldehyde and the convulsion stopped. He left the ward satisfied.

3. What is the dosage of paraldehyde for children?

4. Do you think this treatment was satisfactory?

5. What are the possible causes for the convulsion?

Dr. Fekade happened to pass by the ward the same afternoon. He examined the child carefully and confirmed the chest signs, the high fever, slight drowsiness, and a normal fontanelle. He was unhappy about the convulsion in a sick child with a severe chest infection, so he did a lumbar puncture and then ordered for urgent blood investigations for malaria and a dextrostix test. The blood sugar was in the range of 45-90 mg%; there were no malaria parasites in the blood slide, and the CSF looked cloudy.

6. What diagnosis do you suspect now?

7. What is the treatment?

Treatment was changed to chloramphenicol and penicillin i.v. Next day the CSF results came back from the laboratory: wbc 250/ml (70% poly's); protein 100 mg%; sugar 20 mg%. The WBC in the blood was 12,000.

8. How would you interpret these results?

Despite the more energetic treatment the condition of the child did not improve over the next few days, though the chest signs cleared up and became limited to the right upper lobe. A repeat LP four days later showed a protein increase up to 225 mg% and 335 white cells (68% poly's).

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9. What could this mean?

A subdural effusion was suspected and subdural taps were done, but they produced only little fluid. An Xray of the chest showed consolidations in the right upper lobe with some fine mottling throughout the rest of the lung fields.

10. What is your diagnosis now?

On further questioning of the mother it was found that the child had never been vaccinated with BCG and that the grandmother had been coughing for a long time. A diagnosis of tb meningitis secondary to pulmonary tb with miliary spread was made. Appropriate treatment was started with and the child slowly improved and started to suck. The chest signs disappeared and the temperature settled to normal. A Mantoux test was negative. Twelve weeks later the child was discharged on INH/Thiacetazone. Before discharge the Mantoux test was repeated and was 12 mm.

11. What do you think of the Mantoux test results?

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CASE STUDY 6

One morning Dr. Tesfaye saw at the outpatient department of the regional hospital a 22 years old fisherman with a 5 days history of fever, shivering, headache, and nausea. The man looked toxic, moderately jaundiced, and feverish (38.5°C). The liver was palpable and tender, and the conjunctivae were red and inflamed, but other mucous membranes were pale. He admitted the young man under the diagnosis "fulminant viral hepatitis" and ordered treatment with corticosteroids.

1. What is against the diagnosis of viral hepatitis?

2. What other diseases can cause jaundice and fever?

3. What do you think of the steroid treatment?

After a few days, when he saw the patient again, his condition had deteriorated considerably. The fever continued and the jaundice had deepened. Urine production and blood pressure had fallen, and the man was very drowsy. The thick blood film was negative for malaria and spirochetes; WBC: 20,000 with PNL; liver function tests were abnormal with grossly raised transaminase levels, but only moderately raised alkaline phosphatase; blood creatinine and blood urea were raised as well, indicating renal failure.

4. What diagnosis do you consider now?

5. What is the causative organism and how is the disease transmitted?

6. What might the deteriorating level of consciousness indicate?

7. How would you treat this patient?

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EPIDEMIOLOGY OF SELECTED FEBRILE ILLNESSES IN ETHIOPIA

Dr. Yemane Berhan
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MENINGOCOCCAL MENINGITIS (MAJERAT GETIR OR MOGNE BAGEGN)

Meningitis epidemics occur in a great belt of norther Savannah which extends from Mali across the semiarid Sahel zone sourth of the Sahara into Sudan and norther ethiopia.

Causative Agent: Neisseia meningitidis

Mode of transmission: Airborne, droplets, direct contact.

Reservior of infection: Man

Epidemiology of epidemic meningococcal meningitis in Ethiopia.

- commonly caused by group A & C serotype
- occurs periodically (often in 5-10 years cycles)
- mostly occurs during the dry seasons. The number of cases starts to increase as the temperature rise and reaches peak in March and April. the reseon is during this time people tend to crowd together in a small shelter due to the dry and cold whether.
- are preventable and prdictable
- affects more the younger population, more than 50% of all cases were among children upto 14 years.
- males were slightly more affected than females
- case Fatality Rate (CFR) in recent epidemics was between 2 to 3% on average in various age group (range 2-9%). This was very high before the 1981 epidemic reaching upto 30%.

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Major epidemics in Ethiopia:

1901 - first documented epidemics, then

1935, 1940, 1950, 1964, 1977, 1981-1983 & 1988-1989

Epidemics before 1987 mostly occurred in Northern and Western Ethiopia as far south as Welega. The majority of the reported cases were from Gondar.

The 1988-89 epidemics involved all regions of the country. 45,000 cases and 1,685 deaths were reported at that time.

Factors influencing the invasion of the organism through the mucous membranes or enhance its survival in respiratory droplets are:

- high temperature and low humidity
- herd immunity level of the community
- poor hygienic conditions
- crowding

Preventive and control measures:

- avoid overcrowding
- chemoprophylaxis using rifampicin
- mass immunization

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Surveillance in Ethiopia

There are two types of disease reporting system in the country:

1. Routine : this is the usual monthly report from health institution to the MOH.
2. Emergency/rapid : this type of system is used during the epidemic time in order to facilitate the control activities.

Control activities in Ethiopia

It was poor because :

- poor communication between peripheral and central health service system.
- lack of training of health personnel particularly at periphery
- low health service coverage
- under-reporting and misclassification of diagnosis
- lack of resources : money, material & supplies.

TYPHOID

Causative organism : Salmonella typhi

Mode of transmission : through contaminated food and drinks (oral-faecal route)

Typhoid is one of the first diagnosis in clinics and hospitals whenever the patient presents with fever and a few other non-specific signs and symptoms. The disease is water-borne and a small infecting dose can cause the disease. As many of the water sources in the country are unprotected and open-field defication is common the disease is too common all over the country.

At places where water supply is inadequate the incidence of thyroid rises at the end of the dry season when the scarcity is series. If not treated properly and adequately the disease is fatal.

Prevention :

. Improve the safety(quality) and the amount(quantity) of water supply.

. sanitary disposal of excreta(faeces)

. Identification and treatment of carriers

. Immunization

. Health education

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MALARIA

The disease has been endemic in Ethiopia for many years. The Ethiopian population is concentrated in highlands partly because of the widespread occurrence of malaria in lowlands.

Major epidemic in Ethiopia: The epidemic occurred in 1958. That time 3 million people were affected and 150,000 people died. As a result of this and other influences the malaria eradication service was established in 1959 G.C.

Geographic distribution

- The distribution of malaria is governed by two main factors:
  1. The presence of a suitable mosquito.
  2. A mean temperature high enough to allow the malarial life-cycle to be completed within the mosquito, which is around 20°c.
- three forth of the country's total area is estimated to be malarious.
- two third of the population in the country are at risk of infection.
- prevalence and incidence of malaria has greatly increased since the mid-1980's because of :
  - military campaigns
  - ecological changes
  - population movement, like settlement and villagization
  - deterioration of health services

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. altitudinal effects

. Dega (cold zone > 2,500m) is free of malaria and there is no records of indigenous transmission.

. Weyna-dega (between 1,500-2,500) : malaria is of low endemicity and very unstable with frequent outbreaks resulting in high morbidity and mortality due to low herd immunity level.

. Kolla (< 1,500m) : Malaria is moderately to highly endemic. Malaria in lowlands contributed to overpopulation, over-cultivation and subsequently drought and famine in the highlands; in Ethiopia over 80% of the population live in areas above 1,500m.

Mode of transmission : Mosquito bite and by transfusion of infected blood.

Transmission Periods:

. year round transmission around lakes, swamps, irrigation schemes and large rivers.

. seasonal in Kolla and Weyna-dega zones and peak transmission is after rainy seasons:

. April-May after light rainy season

. September-December after heavy season

Vector : Female anopheles mosquito

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Parasites : There are four species of malaria parasite. Of the four species which cause human malaria Plasmodium falciparum and Plasmodium vivax are the commonest in Ethiopia. Plasmodium malariae and Plasmodium ovale are rarely reported.

Plasmodium falciparum

- comprises 60% of the reported cases
- cause severe, complicated and fatal episodes of malaria
- CFR is about 10% in hospitalized adults and 33% in children less than 12 years of age.

Plasmodium vivax

- widely distributed in Ethiopia (comprises 40% of the reported cases) and often precedes the transmission of P.falciparum.
- more common during the dry season (could be due to relapse)
- it is the major cause of morbidity but not mortality

Prevention and Control

1. Chemoprophylaxis - particularly for children under five years and pregnant women. But there is a fear that widespread use of chloroquine as prophylaxis might favour the spread of resistant strains.
2. Vector control
   - insecticide, DDT, -very expensive
   - avoiding breading sites - involve community
   - applying mesh on the window
   - use of mosquito net
3. Early diagnosis and treatment
4. Health education

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RELAPSING FEVER

Louse-borne relapsing fever (LBRF) remains endemic only in the horn of Africa and Rwanda. It is a disease of poverty, dirt, and disruption.

Causative agent - Borrelia recurrentis (spirochete).

Vector: human head and body louse (Pediculus humanis).

Reservoir : only man.

Mode of transmission : contamination by infected louse body fluids.

Epidemiology in Ethiopia : LBRF is common in the highlands of ethiopia. Several thousand of cases reported annually to the MOH. The reports mainly came from Wello, Shewa, Gondar and Addis Ababa. Most cases of relapsing fever are normally reported during the cool season when the relative humidity is high.

Conditions for transmission:

. susceptible human host

. louse infected with Borrelia recurrentis

. favourable environment : overcrowding, poor hygienic conditions, malnutrition and rainy season. Massive population movement due to settlement or war can cause epidemics of relapsing fever due to poor living conditions and overcrowding.

Prevention and control

. mass Delousing, using DDT

. early detection and management of cases

. improvement of living condition - changing and washing clothes once a week could dramatically reduce the density of lice and the prevalence of the disease.

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TYPHUS [Tessibo Beshita]

Louse-borne typhus is a common cause of acute illness throughout Ethiopia and particularly in the highlands. Although the number of cases reported each year to the MOH has decreased, Ethiopia is still one of the major reporters of the disease worldwide. In the past nearly 90% of the cases reported to the MOH were from central and northern Ethiopia. Epidemics are associated with the cooler climates at higher altitude or with the cold wet season. Poor living conditions and lack of personal hygiene favours transmission.

Causative agent : Rickettesia prowazacki

Vector : human body and head lice (Pediculus humanis)

Mode of transmission : through bite and faeces of the lice.

Reservoir : man only

Prevention and control:

- mass delousing using DDT
- improve personal hygiene.

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TRAINING PROGRAMME ON THE "MANAGEMENT OF ACUTE, FEBRILE ILLNESSES"

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Review Notes

I. Typhoid fever

A wide spread and potentially total infection. The mode of entry is the oral - Faecal route followed by, bacteremia and subsequent affection of many organs.

Etiology

- Classical typhoid fever is caused by Salmonella typhi
  - non capsulated, gram negative bacilli
- Salmonella paratyphi A and B may also produce a similar illness.

Epidemiology

- The organisms gain access to the body thrugh contaminated food and water (usually, contamination is by fecal material)
- Spread is via 5 'Fs": Food, Fluids, Fingers, Fomites, Flies.
- It can cause epidemic disease, especially in developing nations (poor hygiene, unsafe water supply etc.)

Disease Mechanism (Pathogenesis)

- After ingestion the typhoid bacilli invade the intestinal mucosa, grow in its lymphoid tissue, and later enter the blood stream.
- The bacteremia which follows persist until treatment eradicates the bacteroa or they are removed in by the bodys defence mechanism.
- The organism then reinvades the lymphoid tissue in the small bowel, which become the site of inflammation, ulceration, bleeding and perforation.
  - severity depends on the dose of bacteria swallowed.

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Clinical features:

. Incubation Period - 24-48 hrs.
. fever, high grade (38.5 ° C - 39.5 ° C); persistent and headache
. change in bowel habit : diarrhea or constipation
. abdominal cramp, nausea, vomiting.
. dry cough
. change in sensorium
. relative (brady-cardia) slow pulse
. rose spot
. splenomegaly
. meningismus
. other pictures of complications.

--> helpful findings but occur only in some patients

Diagnosis

- Clinical picture + complete blood count plus Blood filn
- Stool, urine and blood cultures.
- Serology (Widal test)
  . rise in titer is more important than absolute value in endemic areas.

Treatment

- Chloramphenicol (the drug of choice); given for 14 days.
- alternatives : Ampicillin (Amoxycillin), Co-trimoxazole,
- General measures (IV fluids etc.)
- for chronic carriers ---> long term ampicillin (Mostly in those infected with bilharziasis)

Complications:

A. **Medical** : Meningitis, Myocarditis, Pneumonia, Osteomyelitis, Arthritis, Orchitis, Typhoid Psychosis, etc.

B. **Surgical**: Intestinal perforation; gastrointestinal bleeding, chronic carrier

Prevention and Control

- water and food hygiene;
- identification and treatment of carriers
- protection of susceptible people e.g. vaccination

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II. Relapsing fevers

Relapsing fevers are a group of acute infections characterized by recurrent cycles of fever which are separated by asymptomatic intervals of apparent recovery.

- Cause: genus **BORRELIA** (spirochaete)
  - Two epidemiologic varieties
    - . Borrelia recurrentis ---> Louse borne relapsing fever
    - . Borrelia duttoni and other borrelia, species -----> Tick - borne relapsing fever

Louse borne relapsing fever

. **Cause** Borelia recurrentis

. **Transmission** By human body and head louse, (pediculus humanus)

. **Epidemiology**
- . Ethiopia is one of the endemic focuses in the world
- . mainly a disease of poverty
- . people affected come usually from prisons; refugee camps etc.

. **Disease Mechanism (Pathogenesis)**

- Inoculation into a human (crashing the lice against the bite site or abrasion caused by scratching) ---> spirochetesa reach the blood stream. These cause fever with accompanying symptoms.

- Clearance of the bacteria by body defense (no fever) ---> another ware of bacteria in the blood stream (fever) --> relapsing fever.

. **Clinical Picture**

. Incubation Period - Variable, but 7-10 days
. High grade fever, headache, malaise, joint pain
. vomiting; abdominal pain
. Nose bleeding, dry cough
. Neck stiffness.

- Examination may reveal hypotension (Low BP), tachycardia, petechial hemorrhage in the skin; right upper quadrant abdominal pain and neck stiffness

- Other findings of complications.

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Laboratory findings:

. Blood film --> positive for the borelia species.
. Other laboratory findings are non specific

Treatment

- The organism is very sensitive to many antibiotic drugs.
- Usual treatment:

  . Low dose penicillin 200,000 - 400,000 Im stat then tetracycline for 5 days
    . In hospital setting
    . Secure Iv line before giving penicillin
    . Vital signs every 10-15 minutes for 1'-2 hrs after the injection
  . Complications of the treatment
    = Jarisch - Herxheimer reaction (JHR)

a) Chill phase - Body t° ; HR ; BP ; RR ; WBC count
b) Flush phase - Peripheral Vaso dilatation
(defer-vescence) - progressive fall in BP ---> shock
  . Pathogenesis largely unknown
  . Rx - control t° ; iv fluid; cardiorespiratory support
    - severe cases are admitted to ICU

Poor prognostic signs

. Bleeding tendency
. Heart failure
. Deep jaundice
. Altered Sensorium

Prevention

. Personal hygiene, shove head
. Control lice - DDT

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III. Typhus

- There are several groups:
  - Endemic typhus (murine typhus) -----> flea borne
  - Epidemic typhus ----------> louse borne
  - Scrub typhus ----------> mite borne
  - etc.

Epidemic (Louse-borne) typhus:

- Several acute febrile illness

Cause: Rickettsia prowazekii, gram negative bacteria

Transmission: Body louse (pediculus humanus corporis)

Clinical picture:

Incubation Period = 5-23 days

- Tachycardia; hypotension
- Fever , high grade, chills, malaise, backache
- very intense head ache, global.
- Skin rash, sometimes gangrenous extremities (vasculitis)
- Tinnitus, vertigo; impaired hearing
- change in sensorium - delirious patient, may lapse into coma.

Diagnosis

- Clinical picture
- Exclude other acute febrile illnesses by appropriate means
- Serological (Weil-Felix) test.

Treatment

- Chloramphenicol 3-5 days
- Alternative: tetracycline (doxycycline)

Prevention

- Eliminate lice
- Personal hygiene

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FEBRILE ILLNESSES IN CHILDREN

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Fever could be present

1. With out localizing sign:- a febrile child with neither an apparent site of infection nor a non infections diagnosis. In most cases additional clinical manifestations will develop within few days and the cause of the fever will be obvious.
2. Fever of unknown origin (FUO):-
   a. a history of fever of >1 wk duration
   b. Fever also documented in the hospital
   c. no apparent diagnosis after an investigation of 1 week in the hospital

* The principal causes of FUO in children are infectious and collagen - vascular diseases. Neoplastic conditions (Lymphoma, Newo blastoma ..) and drug fever should also be considered.

Common Causes of Fever in Children

I. Acute respiratory infections (ARI)
II. Common Central Nervous System infection

a) Meningitis:

- inflammation of the meninges which is the covering of the brain.
- could be due to pyogenic bacteria, viral or tuberculosis.
- The clinical manifestations of pyogenic meningitis during infancy:-
  - signs of meningeal inflammation may be minimal; only irritability, restlessness, and poor feeding may be noted; fever is usually present.
  - nausea, vomiting, anorexia, nuchal rigidity, occasional photophobia, bulging fontanel in the infant and headache in older children.
  - Seizure, stupor, coma and focal neurologic signs may be present. Sub-dural effusion may also occur.

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- Diagnosis:- do lumbar-puncture (LP) and analyze the cerebro spinal fluid (CSF).

- Treatment:- Immediate administration of appropriate IV - antibiotic for at least 10 days

III. Bacterial Infections

a) Septicemia is the presence of bacteria in the blood associated with active disease, localized or systemic.

. a clinical diagnosis of presumptive septicemia should be made when fever and general appearance of the patient suggest serious illness.

. appropriate cultures (Blood, Urine, CSF.) should be obtained and antibiotic therapy should be provided promptly by the intra venous route.

b) Osteomyelitis :- bacterial infections of bones

- Clinical manifestation of acute osteomyelitis

. most often between 3 and 12 yrs
. most commonly caused by staphylococcus aureus
. fever, may have systemic signs of toxicity
. infection localizes most often in the long bones
. swelling, erythema, tenderness, and decreased movement of the involved part
. elevated white blood cell count (WBC) erythrocyte sedimentation rate (ESR), and X-ray finding of bone disease.

- Treatment :- Intra venous administration of appropriate antibiotic for 4-6 wks or longer, as an in-patient.

c) Septic arthritis :- bacterial infections of joints

- Clinical manifestations:-

. most commonly during the 1st year of life.
. onset may be sudden with systemic symptoms and fever.
. there will be swelling, pain, erythema of the overlying skin, tenderness, warmth, and limitation of movement of the affected joint. Limping is present.
. elevation of the WBC count and ESR

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- Treatment:-

  . patient should be admitted to a hospital surgical drainage - for hip joint and shoulder joint arthritis
  . appropriate intra venous antibiotic for 14-21 days.

d) Urinary tract infection (UTI)

- Clinical manifestations:-

  . In-infancy :- fever, weight loss, failure to thrive, nausea, vomiting are common.
  . Later - in child hood :- urinary frequency, pain during micturition, Urinary in continence, urgency, bed wetting in a previously dry child, abdominal pain, and foul-smelling urine are common symptoms.
  . chills and flank pain and tenderness may be present in acute pyelonephritis.
  . U/A - will show pyuria (pus cells >5/HPF) and urine culture may grow the organism.

- Treatment:

- appropriate antibiotic
- if there is recurrent UTI, investigate the patient to rule out anatomic abnormalities of the urinary tract.

e) Typhoid fever:

- Clinical manifestations

  . In-infants:- vomiting, abdominal distention, and diarrhea are common. Fever, seizure, hepatomegaly, gourde, anorexia and weight loss may occur.

  . In older children:- Fever, malaise, lethargy, myalgia, headache, abdominal pain and tenderness, diarrhoea, epistaxis and cough may occur. Delirium, stupor, hepato-splenomegaly and rhonchi may be present.

  . A macular (rose spots or maculo popular rash may be observed.

  . Cultures of blood, urine, and stool may grow the organism.

  . Serology test (widal) is positive in most cases

- Treatment:- chloramphenicol 50-100 mg/kg/24 hrs divided in 4 doses Po or IV, for 10-14 days.

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f) Relapsing fever

- Clinical manifestations:-

  . Sudden on set of high fever, head ache, photophobia, nausea, vomiting, myalgia, and arthralgia.

  . bleeding manifestations (epistaxis, hemoptysis etc.) are common

  . A diffuse, erythematous, macular, and/or petechial rash over the trunk and shoulder is common.

  . Lympdenopathy, splenomegaly, hepatomegaly, jaundice, CNS manifestations also occur.

  . Patients will have episodes of remission of symptoms and then relapse (re-occurrence) of the febrile episodes.

  . Diagnosis:- demonstration of the organism on blood smear.

- Treatment:- ideally, patients should be hospitalized.

  . start intra venous fluid, resuscitate the patient
  . administer appropriate antibiotics. (procaine penicillin, chloramphenicol, tetracycline, erythromycin)
  . follow the patient closely for signs of Jarisch-Herxheimer reaction (tachycardia, tachypnea, hypotension, shock --> patients may die)
  . follow the patients for signs and symptoms of relapse.
  . advise on good personal hygiene and delousing of persons, dwellings and clothing with insecticides (DDT).

IV. Viral infections

a) Measles

- Clinical manifestations

1. an incubation stage of about 10-12 days, usually with out any symptoms.

2. The prodromal stage which lasts 3-5 days, with fever, cough, rhinitis, conjunctivitis. Koplik spots are grayish white dots, on the buccal and pharyngeal mucosa.

3. a final stage with a maculopapular rash erupting successively over the neck and face, body, arms, and legs with in 2-3 days, accompanied by high fever. Then the rash begin to fade starting on the face down wards.

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Treatment:- antipyretics, bed rest, adequate fluid in take avoid strong light during the period of photophobia. Antibiotics if it is complicated by otitis media and pneumonia.

b) Herpes simplex

- Clinical manifestation:- the viruses characteristically produce a vesicular lesion (aggregates of thin walled, clear fluid containing vesicle) on an erythematous base. These rupture, scab, and heal within 7-10 days without leaving a scar.

c) Varicella (chicken pox)

- Clinical manifestations:-
  - Peak incidence is 5-9 yrs.
  - fever, malaise, anorexia usually precede the rash by 24 hrs.
  - The characteristic rash begins as crops of small, red papules which almost immediately develop in to clear, often oval "tear-drop" vesicles on an erythematous base; they are usually not umbilicated. The contents usually become cloudy within about 24 hrs. When the vesicles are easily broken and become scabbed. Vesicles continue to appear for 3-4 days. Starting on the trunk, they spread to the face and scalp. Characteristically, the eruption consists of papules, early and late vesicles, and crusts present at the same time. Itching is also present.

- Treatment:- Symptomatic

d) Herpes Zoster

  - Usually starts with malaise, fever, and pain along the involved dermatome.
  - then groups of red papules appear, distributed along a dermatome. The individual lesions quickly vesiculate, become pustular, dry up and scab in the course of 5-10 days.

- Treatment:- Symptomatic :- antipyretic, analgesics.
  - Soaks and calamine or other drying lotions may be helpful. Antiviral drugs could be given.

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e) Mumps:

. Fever, muscular pain, headache, malaise
. Usually characterized by pain and swelling in one or both parotid glands. Swelling slowly subsides within 3-7 days but occasionally lasts longer.

- Treatment:- Symptomatic - bed rest, analgesics, antipyretics.

f) Hepatitis

- Clinical manifestations

. fever, malaise, nausea, vomiting, anorexia, right upper quadrant pain, jaundice are usual signs and symptoms of hepatitis - A (Infectious hepatitis).
. In Hepatitis - B (Serum hepatitis), there may be arthralgia, or skin rashes, the liver is usually enlarged and is tender, splenomegaly and lymphadenopathy are common.

- Treatment:- Supportive

. diet low in fat is more acceptable
. in serve anorexia or emesis, give IV fluids.

V. Rickettsiae

a) Louse-Borne-Typhus:

. etiology:- Rickettsia Prowazeki :- small, fastidious, coccobacillary organism

- Clinical manifestations:-

. Disease begins with headache, fever, anorexia, and restlessness. Discrete, pale, rose-red macules or maculopapules appear 1-5 days after the onset of illness; intense myalgia, malaise and splenomegaly occurs. Symptoms of central nervous system affection, myocarditis, and renal involvement are more severe manifestations. Serologic test (weil-felix) is positive.

- Treatment:-

. Chloramphenicol 50-100 mg/kg/24 hrs divided into 4 doses for 7-10 days.

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VI. Protozoan Infection

a) Malaria

- Etiology and route of infection:-
  - Plasmodium falciparum (the more severe form), P. vivax, P.Ovale, and P. Malariae are the four etiologic agents.
  - Malaria is usually acquired from the bites of previously infected female anopheline mosquitoes.
  - In other instances, it has developed following transplacental passage or after the transfusion of infected blood.
- Clinical manifestations:-
  - Behavioral changes such as frightfulness, anorexia, unusual crying, drowsiness, or disturbance of sleep may be observed. Fever, chills, headache, nausea, generalized aching, splenomegaly, hepatomegaly, and jaundice.
  - In severe falciparum malaria, there may be manifestations related to the cerebral, pulmonary, intestinal or urinary system.
- Diagnosis:- Identification of parasites in the blood smear.
- Treatment:-
  - Supportive treatment and management of complications.
  - Specific chemotherapy:- antimalarial drugs
    - Chloroquine 10 mg/kg po start and then
      5 mg/kg after 6 hrs and then
      5 mg/kg for the next 2 days, daily.
  - For resistant malaria
    - Quinine
    - Fansidar.

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CASE MANAGEMENT OF ACUTE RESPIRATORY TRACT INFECTIONS IN CHILDREN

Acute respiratory infections (ARI), diarrhoea, and malnutrition are the most common causes of illness and death in children in developing countries together with malaria in endemic malarious areas.

One quarter to one third of deaths in underfive children in developing countries are attributed to ARI. ARIs are not only a leading cause of death, they are also occurring frequently. It is estimated that a child in an urban area has five to eight episodes of respiratory infections annually during the first five years of life and a child in a rural area one to three episodes per year. ARIs are also a leading reason for the use of health services. They usually constitute 22-42 % of children attending outpatient services. Most of them suffer of upper respiratory tract infections (70-80%).

Of ARIs pneumonia is the most common cause of death among children under five years of age. In one study 79% of deaths from ARI are attributed to pneumonia. Bronchiolitis, obstructive laryngitis, and epiglottitis are other fatal conditions.

In developing countries mortality from ARI is 30-70 times higher than in developed countries. In developing countries the case fatality rate of pneumonia as obtained from hospital figures ranges from 5-10 %; in rural areas without adequate health care facilities it is estimated to be over 10%. Particularly infants are at risk since the mortality in the age group below 1 year is four times higher than in the 1-4 years age group. It is found that in developing countries bacterial pathogens play a far greater role in lower respiratory infections than in developed countries, which contributes to the higher mortality. Also childhood malnutrition contributes to increased mortality.

In order to decrease the mortality from ARIs it is important that health workers know how to differentiate pneumonia from upper respiratory tract infections, and treat pneumonia promptly with antibiotics.

HOW TO DIAGNOSE PNEUMONIA

Most children with pneumonia present with cough, but most children with cough do not need antibiotics. Therefore we have to look for other signs and symptoms to diagnose pneumonia.

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Rapid breathing (Tachypnoea) is the most useful sign to diagnose pneumonia. Rapid breathing is defined as more than 50 breaths per minute. While counting respirations, the child should be awake and alert. In addition to rapid breathing, a child with pneumonia may show nasal flaring, inter costal chest indrawing, grunting, cyanosis, or failure to feed, as signs of more severe pneumonia.

Fever or the presence of purulent sputum are no good signs to diagnose pneumonia in young children, while crackles (crepitations) on auscultation of the chest are difficult to hear in small children particularly for PHC workers.

MANAGEMENT OF PNEUMONIA IN CHILDREN

1. Mild pneumonia
Children with mild pneumonia (cough and tachypnoea, but no chest indrawing) can be treated as an out-patient with either oral antibiotics (cotrimoxazole, amoxycilline, ampicilline) or daily injections of procaine penicillin for five days. In infants below 2 months of age it is advisable to add gentamycin.

2. Moderate pneumonia
Children with pneumonia who besides rapid breathing have chest indrawing preferably ought to be admitted for more intensive treatment with 6 hourly injections of benzyl penicillin or ampicillin.

3. Severe pneumonia
Children with pneumonia who have chest indrawing and either cyanosis or are too sick to feed, are at a high risk of dying and should be admitted for treatment with 6 hourly injections of benzyl penicillin and in addition chloramphenicol when the child is over two months of age. Chloramphenicol is given i.m. 6 hourly until the child starts to recover (usually after 2-3 days) , there after orally. Gentamycin can be given in stead of chloramphenicol in children below two months of age. When cyanosisis present the child needs oxygen by intra-nasal catheter at 0.5 litre/minute. Feeding by naso-gastric tube may be necessary. Gently sucking out the nose is indicated when blockage by mucous causes respiratory distress.

Benzathine penicillin is not recommended for the treatment of pneumonia.

Amoxycillin is better absorbed than ampicillin and gastro-intestinal side effects are slightly less common, while medication only needs to be given 8 hourly.

Medicines such as expectorants, cough suppressants, mucolytics, decongestants, and antihistamines are expensive and ineffective in pneumonia and might even be harmful.

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DOSAGES OF ANTIBIOTICS

Procaine penicillin:	50,000 U/kg i.m. once each day.
Amoxycillin:	15 mg/kg/dose every 8 hours.
Ampicillin:	12.5 mg/kg/dose every 6 hours.
Cotrimoxazole:	sulphamethoxazole 20 mg/kg and trimethoprim 4 mg/kg per dose orally 12 hourly.
Benzyl penicillin:	25,000 - 40,000 U/kg/dose i.m. or i.v. every 6 hours.
Chloramphenicol:	25 mg/kg/dose every 6 hours.
Gentamycin:	2 mg/kg/dose i.m. every 8 hours.

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Lower Respiratory Tract Infectious in Adults

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Although respiratory tract infections are very common in general out-patient practice true "pneumonia" is relatively uncommon.

Disease mechanism (pathogenesis)

- Colonisation of the oropharynx by " respiratory" pathogens
- Inhalation of bacterial pathogens
- Alveolar filing - consolidation

Clinical Evaluation

The setting of the clinical circumstance is very useful in the evaluation of a patient with cough, fever and chest pain.

- ? Where; community acquired
  hospital acquired
- When, after epilepsy ---
  ? Aspiration
- Clinical presentation
  Viral like - "Atypical Pneumonia"
  Bacterial like - "Lobar Pneumonia"
- Underlying Host factors
  Alcholism
  Chronic bronchitis

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Table 1. Classification and Treatment of Pneumonia

Underlying condition	Usual pathogen/s	Presumptive therapy
Community acquired pneumonia	Pneumococcus	Penicillin G or Ampicillin
Atypical pneumonia	Mycoplasma, Chlamydia	Erythromycin
Alcoholism	Pneumococcus, Klebsiella Staph. aureus	Cephalosporin with Haemophilus influenza activity (i.e. 2nd or 3rd generation)
Chronic bronchitis	Pneumococcus Haemophillus influenza Branhamella Catarralis	Ampicillin or Cephalosprin with H. flu activity
Aspiration pneumonia	Mouth anaerobes	Penicillin G or Clindamycin
Hospital acquired pneumonia	Gram negative bacilli	3rd generation cephalopporin plus amingoglycoside

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Treatment of Tuberculosis

About 20 million people world-wide are suffering from tuberculosis. The impact of HIV infection on tuberculosis control programme has been devastating. The availability of new and potent drugs, particularly rifampicin has made it possible for patients to be treated for a short time. Short-course chemotherapy has several advantages over "standard" treatment. These include better patient compliance and thus achievement of higher cure rates. Less development of resistance organisms. However, cost and availability of drugs are important considerations.

Table 2. Recommendations for the treatment of tuberculosis

	Drugs	Duration of Therapy	Cost
Standard treatment	Streptomycin plus TB450 (INH 300 mg thiacetazone 150 mg)	2 months	25.00 Br.
		12-18 months	14.00 Br.
	Streptomycin (1 gm/day)/or Ethambutol (15 mg/kg/day)	2 months	25.00 Br.
	Pyrazinamide (25 mg/kg/day) plus INH (300 mg/d Rifampicin (600 mg/d)	9 months	9.00 Br. 385.00 Br.

Shorter courses of 6 months and intermittent twice weekly supervised. Treatment for 9 months (INH 900 mg. and Rifampicin 600 mg) have also been employed with encouraging results. Short course chemotherapy have also been shown to be effective for the Treatment of extra pulmonary tuberculosis.

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CHRISTIAN RELIEF & DEVELOPMENT ASSOCIATION

TRAINING PROGRAMME ON THE "MANAGEMENT OF ACUTE, FEBRILE ILLNESSES"

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Review Notes

I. Meningitis

Definition

Meningitis is inflammation of the membranes (leptomeninges) covering the brain and spinal cord. Based on the causative organisms meningitis is broadly divided into:

1. Acute bacterial (pyogenic) meningitis
2. Aseptic Meningitis (viral)

Causative Organisms

The causes of bacterial meningitis are different in different age groups. However the three most common causes of bacterial meningitis in both adults and children are:-

1. Hemophilus Influenzae
2. Neisseria Meningitids
3. Streptococcus Pneumoniae

Epidemiology

Meningitis caused by the common bacterial pathogens occur sporadically throughout the year. Meningococcal meningitis however causes large epidemics in countries that lie within the "African Meningitis Belt" including Ethiopia (see map below). It affects mainly children and young adults. Epidemics occur in the hot dry season between January to the end of June. several epidemics have occurred in Ethiopia. The latest one in 1992-93 affecting 4,000 people.

Disease Mechanism (Pathogenesis)

In most instances bacteria that cause meningitis first "colonize" the oropharynx. Later they invade the blood stream before they finally reach the meninges and produce an inflammatory response in

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the Cerebro Spinal Fluid (CSF). In some clinical situations they may directly reach the central nervous system from nearby infections e.g. middle ear infection (Otitis media)

Clinical Presentation

Most of the clinically observed symptoms and signs are a direct consequence of infection in an anatomically very confined space i.e. the brain and spinal cord. Systemic complications like shock and skin lesions may also be present. figure one shows the common signs and symptom of bacterial meningitis.

Mode of Diagnosis

Diagnosis is made on the basis of the clinical presentation and laboratory results. The most important procedure in patient & with bacterial meningitis is lumbar puncture (LP) to obtain CSF for laboratory analysis. This is usually done by doctors but under epidemic situations may be performed by specially trained nurses.

Cerebrospinal Fluid changes in patients with bacterial meningitis.

Colour	turbid - pus
Pressure	↑↑
Cell count	↑↑ (>500 cells/mm^3)
Protein	↑↑ (> 45 mg/dl)
Sugar	↑↑ (< 2/3 of blood sugar)

Complications

Both local neurological and systemic complications occur in patients with bacterial meningitis. These include:

- Deafness
- Blindness
- Hemiplegia
- Arthritis
- Blistering Skin Lesions
- Shock

Management

Care of patients with bacterial meningitis include both supportive and definitive measures.

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General Management

- Correction of fluid and electrolyte disturbances careful monitor and administration of intravenous fluids D/W, D/S
- Control of high fever - tepid sponging, fanning, ASA, paracetamol
- Nursing of comatose patient - lateral position, frequent suctioning

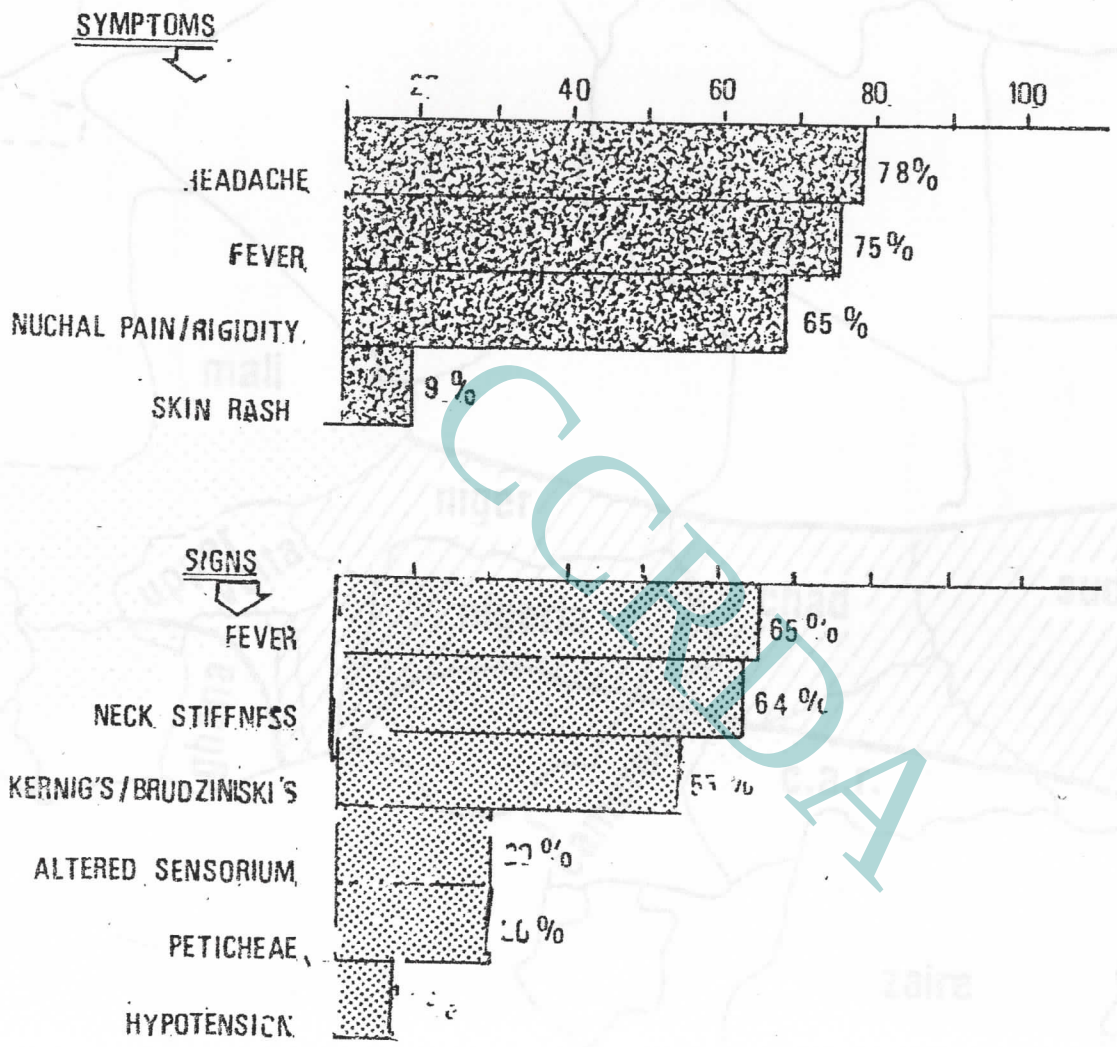
Antimicrobial Agents for the treatment of Bacterial Meningitis

Organism	Drug of Choice	Alternative Drug	Duration of Therapy
S. Pneumonia	Penicillin G	Chloramphenicol third-generation Cephalosporin	10 - 14 days
H. Influenza	Chloramphenicol	Third-generation Cephalosporin	10 - 14 days
N. Meningitides	Penicillin G	Chloramphenicol or fluid-generation Cephalosporin	7 - 10 days

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Clinical Features

Fig 3 SYMPTOMS AND SIGNS ON ADMISSION



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Non-infections causes of fever

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- Fever is not always due to infection with infection.
- Several non infections disease states are accompanied by fever.

I. Neoplasms

A. Solid, localized tumors (Kidney, Lung, Pancreas, Liver, etc.)
B. Metastatic tumors (from GI Lung, Melanoma, Female genital tract)
C. Tumors of the immune system; (Lymphoma, Leukaemia)

II. Connective tissue disease

A. Rheumatic fever
B. Systemic Le ıpus Erythematosus
C. Rheumatoid Arthritis
D. Others (Hypersensitivity vasculitis; P.N., W.gr.)

III. Granulomatous disease

A. Sarcoidosis
B. Crohns disease; Ulcerative colitis
C. Erythme nodosum

IV. Miscellaneous

A. Drug fever
B. Pulmonary emboli
C. Thyroiditis
D. Hemolytic States
E. Hematoma

V. Thermoregulatory disorders

Central -----> brain tumors, stroke (CUA) encephalitis
Peripheral --> hyperthyroidism; Pheochromocytoma

VI. Psychogenic fevers

A. Factitious fever

VII. Undiagnosed

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EPIDEMIOLOGY OF MALARIA

INTRODUCTION:

- Indigenous malaria has been recorded as far as 64° N & 32 ° S latitude, 400 m below sea level and 2800 m above sea level.

- In Ethiopia, any place lying below 2000 m in sea level is considered as potentially malarious area. This implies that 75% of the land, and 64% of the population are in the malarious areas.

- The major species of malaria parasite in the country are plasmodium falciparum and plasmodium vivax.

  Plasmodium malariae is very rare and P.ovale is practically non-existent in Ethiopia.

Types of transmission:

Natural - Infection by bites of infective anopheles mosquito source of infection being sick or symptomless person.

Accidental - Unintentionally infecting a person. Eg. commonly blood transfusion, using the same needle for injections.

Congenital - Newborns getting the infection from their mother. very rare but it occurs.

Deliberate - Experimental trials of antimalarial drugs.

- In studying the epidemiology of malaria it would be simpler to consider 3 components of the epidemiological triangle the host, the agent & the environment.

HOST

AGENT ENVIRONMENT

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HOST factors

Defn. . The host is the human being who is suffering from malaria.

. Age & sex are not important but children have higher degree of susceptibility.

. Some factors in the blood provide protection against infections with malaria parasite. Eg. Haemoglobin S and F absence of Duffy blood group determinants - Plasmodium Vivax, Glucose 6 Phosphatse Deficiency (G6PD) deficiency - from malaria infection

. In endemic areas a proportion of the population is carrier of gametocytes.

AGENT factors:

Defn. . The agent is the organism which is responsible for the disease. In malaria, it is the plasmodia and the anopheles.

. Some species of anopheles are insusceptible to certain species and strains of human plasmodia.

. The pattern of the relapses differ with the strains of Plasmodium vivax.

. There are several stains of plasmodia with different degrees of resistance to antimalarial drugs.

. The number of Anopheles species must be adequate and should be present in or near human residences to be effective vectors.

. Life time of the vector is important

ENVIRONMENT factors:

. Climate - Temprature & Humidity affect
- life cycle of the mosquito
- dev't of the parasites in the mosquito
- life time of mosquito

- Strong Winds
- flight of Anopheles,
- egg laying

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- Rainfall
  - may increase relative humidity higher probability of survival of Anopheles.
  - may negatively affect transmission

. Biological factors
  - presence of water bearing plants
  - presence of cattle

. Socio-economic factors
  - Sanitation
  - Housing
  - occupation
  - poverty etc.

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CLINICAL PICTURES OF MALARIA

- Incubation period : 10-15 days or more.
- Primary fever
  - release of merozoites (RBC)
  - irregular (primary fever) fever.
  - typical attack 3 stages

  Cold stage
  - 15 min. - 3 hrs.
  - weariness, headache, nausea, chills

  Hot stage
  - dry & hot skin
  - full pulse & perspiration
  - 30 min - 6

  Sweating stage
  - profuse sweating
  - cold & moist skin
  - puse
  - Pf. feels weak and falls asleep

  * This continues every 48-72 hrs.
  * In P.f more severe.

- Short-term recrudeseenes of fever
  - In partially Rxed pts.
  - In semi-immune pts
  - Less severe than the 1° attack

DIAGNOSIS

- Definitive Dx - parasite in the blood
- Clinical Signs & symptoms
  - mostly
  - confusion & other diseases (40-50%)
  - climinate other possible diseases
- Dx at peripheral level
  - community posts without lab.
  - CHW's limited training
  - Dx by signs & symptoms only
  - Few patients with Sign/symptoms of severe & complicated malaria
  - Children < 5 yrs with fever --> Treatments as a case of malaria + other possible Dx.
  - Patients with no response to initial Rx Refer with treatment

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- Diagnosis at intermediate level & lab.

  - No lab. but able to administer Intramuscular subcutaneous drugs
  - Better training
  - Diagnosis by Sign/symptoms
  - More detailed clinical assessment possible
  - Better possibility of diagnosis of other diseases
  - Attends <u>new</u> , <u>referred</u> & <u>follow-up on Rx</u>

- Diagnosis at Intermediate level with lab
  - same as the above
  - able to - confirm Diagnosis
    - monitor treatment response

- Dx at hospital level

  - In addition to the above:-
    . better qualified personnel
    . better facilities of Dx and management of severe and complicated malaria cases
    . Rx of many more diseases possible
    . All patients admitted should be assessed by microscopy before & after treatment.

MALARIA TREATMENT

- Major Aim
  - Shorten duration
  - Prevent severe malaria
  - Prevent death

- Drug of Choice
  . Chloroquine - 10 mg/kg 1st day Per OS (by mouth)
  - 10 " 2nd " " "
  - 5 " 3rd " " "

  . If there is vomiting give 1.M injections. Chloroquine 3.5 mg base/kg every 6 hrs to a total dose of 25 mg/kg. (change to oral route as soon as possible)

- Second Line Drugs

  . Fansidar
  . Quinine
  . Other drugs

- Advise the patient (or Ask)
  (TO) - Stay for 1 hr. After 1st dose
  - Come back for Rx/If no response
  - If Rxed for the same illness somewhere else.

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MANAGEMENT OF MALARIA IN PREGNANCY AND CHILDREN

In pregnancy (Why special attention)

- P.f carries high mortality to the foetus + the mother (100 communal immunity)
- High risk
- Therefore, prevention of infection vital & essential
- Advise prophylaxis
- Do blood film exam.
- Rx in any case (+ve/-ve)
- Chloroquine prophylaxis every wk 300 mg base 6/52 after delivery.
- Additional protective measures (Bed net for the baby)
- If malaria develops despite prophylaxis/Rx --> Quinine
- Fansidar !! weigh risk

IN CHILDREN

Why?

- Children in the 1-5 yr age group not yet developed immunity against malaria.
- For this reason are more susceptible to frequent and severe type of infection.
- S/sx are non-specific:- can be miss diagnosed !
- Therefore, any child with fever should be treated for malaria! + any other disease suspected.

REMEMBER!

- No Fancidar 1yr of age!
- In pregnancy "Fansidar!!" should not be used. But weigh the risk !!!

MANAGEMENT OF SEVERE AND COMPLICATED MALARIA

MANIFESTATION

1. Neurological
   - altered/consciousness
   - convulsions
2. Persistent vomiting
3. Very high temperature (39 ° C and higher & dry skin)
4. Very low temperature (< 35.7 ° C/)
5. Anaemia severe

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6. Renal failure - very small/no urine
7. Pulmonary oedema - difficulty in breathing & cough
8. Circulatory collapse (shock) - Feeble, rapid pulse & cold limbs
9. Spontaneous bleeding
10. Hemoglobinuria - black urine
11. Jaundice - yellow coloration of the eyes
12. Pregnancy with fever
13. Failure to respond to Rx within 2-3 days
14. Reaction to drugs - interfering with normal daily activity
15. Conditions that cannot be managed locally.

CONDITIONS THAT NEED REFERRAL

- Peripheral --> intermediate level
  - Repeated vomiting & inability to retain medication
  - Inability to eat/drink
  - Failure to respond to initial Rx

- To maximum care unit
  - Difficulty in walking, sitting up, standing up & walking without obvious cause
  - Unexplained heavy bleeding.
  - Passage of no or little urine /dark urine/
  - Change in behaviour (confusion, drowsiness)
  - Altered consciousness or comd
  - Convulsions
  - Jaundice and/or anaemia
  - Shock/circulatory collapse
  - Difficulty in breathing.

MANAGEMENT

- **INTERMEDIATE LEVEL**

  - should be within reasonable distance
  - can have difficulty to manage severe cases
    eg. - Lack of adequate training
    - Absence of alternative drugs, equipment.
  - should at least give I.M/S.c antimalarial drug and refer the patient to the next level.

- **HOSPITAL LEVEL**

  - has qualified personnel to Rx severe cases
  - adequate facilities are essential
  - lab. confirmation vital
  - I.V. treatment should be available
  - Other tests to role out/Dx other diseases are essential Eg. L.P

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CHLOROQUINE SENSITIVE	CHLOROQUINE SENSITIVITY UNKNOWN
PERIPHERAL LEVEL	
CHLOROQUINE tab/syrup 10 mg base/kg THEN refer/continue 5 mg base/kg at 6, 24 & 48 hrs 25 mg/kg	QUININE : tab. by mouth 10 mg salt/kg (if available) THEN refer (if not continue 10 mg salt/kg every 8 hrs for 7 days
INTERMEDIATE LEVEL	
CHLOROQUINE: 3.5 mg base/kg 9.6 hr. 1.M/s.c P.O Total - 25 mg base/kg If any problem refer! OR Quinine	QUININE: - 20 mg salt/kg - 10 adding dose 1.M - 10 mg/kg 8hr - until pt. can swallow 7 days If any problem refer!
MAXIMUM POSSIBLE HEALTH CARE	
CHLOROQUINE:- - 5mg base/kg In isotonic fluid constant l.v. infusion over 6 hrs. - Repeat every 6hr. 5 doses OR Quinine	QUININE :- - 20mg salt/kg (10 adding dose) by infusion over 4 hours in 5% D/S (5-10 ml/kg body wt depending on the fluid balance - Repeat 9 8-12 hrs. until patient can take oral medication

TREATMENT FAILURES

REASONS:-

- inadequate Rx (vomiting, not taking Rx)
- fever due to other diseases
- poor quality of drug (Exp. date)
- drug vomited
- resistant parasite

Therefore patient returning & Sxs after Rx ? Resistant parasitic

1. Confirm Dx of malaria
   Lab. Dx Refer (if lab. not available)
2. If no Lab. give alternative drug (worse clinical sxs)
3. Alternative drugs should be available at specified level.
4. Remember incomplete Rx Dev't of resistance. Therefore active complete Rx as far as possible.

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IMPORTANCE OF REFERRAL FACILITIES

- Diagnosis & Treatment of severe & complicated cases
- Technical guidance
- Basic training, retraining & supervision
- Supplying drugs
- Support for evaluation of services provided and the status of the local malaria situation.

CONTROL OF MALARIA OUT-BREAK

DEFINITIONS

ENDEMIC MALARIA - Constant presence of malaria in an area.
EPIDEMIC MALARIA - Cases clearly in excess of the expected number for a given population at a particular place & time.

How can we know that there is an out-break?
. Establish the seasonal trend of the disease

How can we determine the trend?
. Compile your health institution data (4-5 yrs)
. Analyse your health institution data

How can we tell whether there is an epidemic?
. Compare the out-break data with the compiled data.
. Any unusual increase OUT-BREAK

Where do epidemics occur?

. in areas with low malaria endemicity
. endemicity through vector control measures
. where there is large population movement
. in areas where there was no transmission
  . absent in recent post/
  . fringe areas

When do epidemics occur?

. Any time of the year. (If conditions are fulfilled)
. Usually following - small & big rains.

Some indicators of a possible out-break

. increased attendance at health
. " demand for anti-malarial drugs
. " rate of absenteeism at schools / work places.
. " in the No sick people/deaths in the community.
. febrile cases among migrant workers engaged in dev't areas
. in the no of mosquitos in a locality.

ANY OF THE ABOVE IMPLIES POSSIBLE EPIDEMIC

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PRECIPITATING FACTORS

- Abnormal rainfall low or high
- Development activities Eg. Dams, irrigation etc.
- Insecticide resistance
- Interruption of vector control measures
- Drug resistance
- Population movement
- Lack of early diagnosis & Rx facilities

MEASURES TO BE TAKEN IN EPIDEMIC CONTROL

1. CHEMOTHERAPY
   - Schizonticide
   - Gametocytocide

2. LARVAL CONTROL
   - Env. control
   - Chemical
   - Biological BTI, larvivourous fish

3. ADULT MOSQUITO CONTROL
   - Chemical
   - Zooprophylaxis
   - Housing site

INFORMATION (Possible epid.) ← Review routine information
Surveillance, clinical cases
Community information

CONFIRMATION

DETERMINE WHETHER IT IS EPIDEMIC ← Check records & seasonal incidence

TREAT CASES/POSSIBLE CASES

→ IDENTITY CAUSE FOR THE EPIDEMIC

ATTACH SOURCE & TRANSMISSION

INSTITUTE PREVENTION

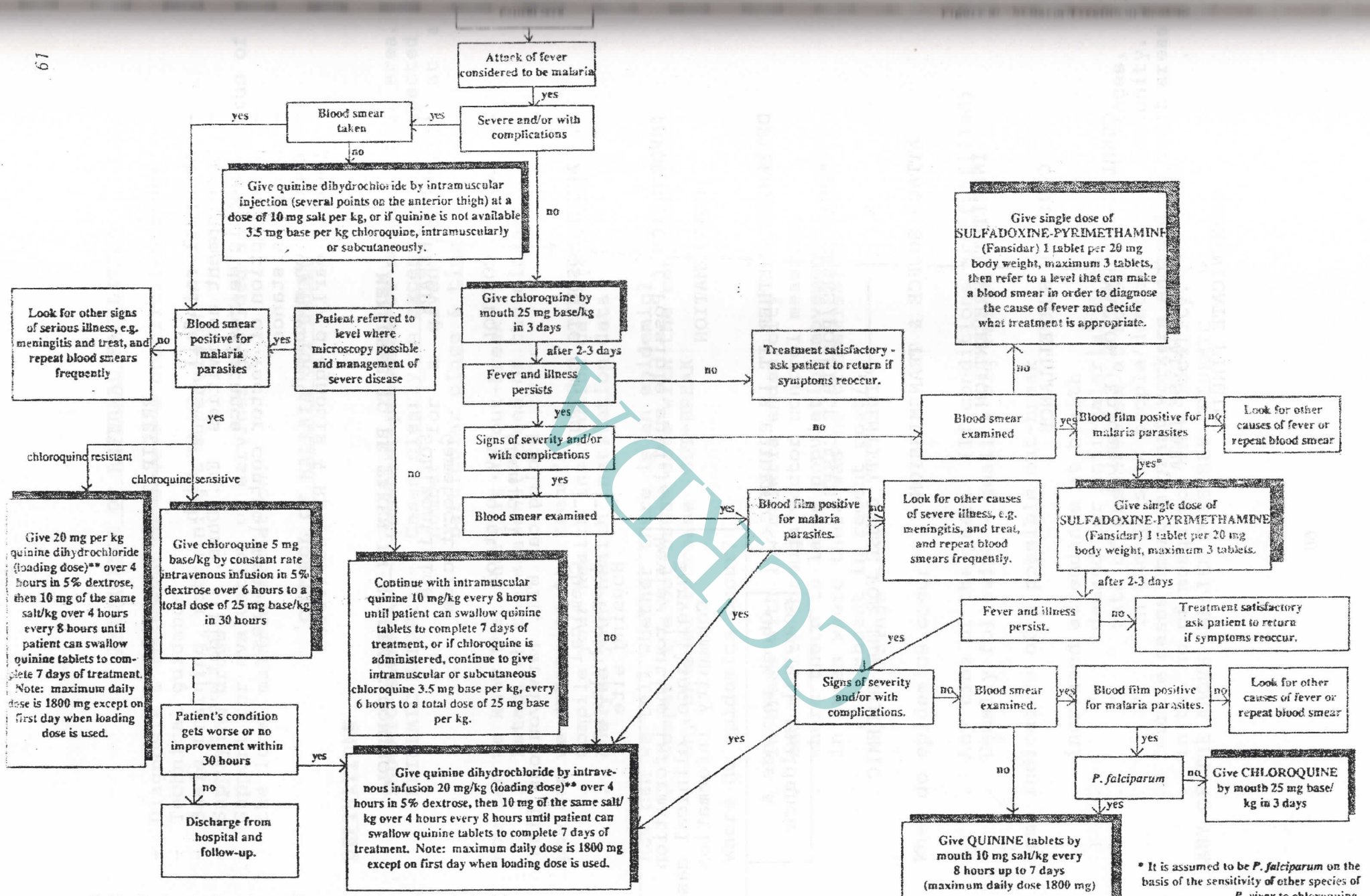
CONTINUE SURVEILLANCE

COMPILE INFORMATION (Epidemic)

PROCESS AND ANALYSE DATA

COMMUNICATE FINDING

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** A loading dose should not be used if the patient has already received quinine or quinidine within the preceding 12-24 hours, or mefloquine within the previous 7 days.

* It is assumed to be *P. falciparum* on the basis of the sensitivity of other species of *P. vivax* to chloroquine.

CDR

August 11, 1993
Kasahun Temesgen M.D

HIV/AIDS

As of June 1993 718, 894 cumulative AIDS cases in adults and children have been reported to WHO. WHO estimates that over 2.5 million cumulative AIDS Case have occurred to date 80% of all estimated AIDS Cases in developing countries.

Estimated 14 million people have been infected by HIV the human immunodeficiency virus which causes AIDS. This figure is estimated to reach 30-40 million people by the year 2000. Out of 14 million-8 million people are in sub-saharan Africa (the worst affected area).

In Ethiopia up to the end of June 1993 a total of 6726 AIDS Cases were reported. There is a problem of under diagnosis and under reporting otherwise the DAC based of sero prevalence data estimates there are more than 30,000 AIDS cases to date.

Summary Reported AIDS Cases

Table 1. Summary of reported AIDS Cases in Ethiopia as of 30th June 1993.

Variable	Cumulative value
Case Definition Criteria	WHO case definition with serology
Number of AIDS Cases report	6,726
Number of reporting hospitals	47
Average (mean) delay in reporting	2 months
Proportion of paediatric cases (age < 15 yrs.)	1.5%
Average age of patients	30.2 years
Sexually active age group (15-49 yrs.)	93.8%
Proportion of married people	33.4%
Proportion of elderly (age > 65 yrs.)	0.2%
Male to Female ratio of total cases	1:7:1
Heterosexual transmission	84.1%
Patients residing in Addis Ababa	40.3%
Proportion of reported cases by A.A hospitals	51.7%
Major clinical symptoms	
Weight Loss of >10% body weight	92.9%
Prolonged Fever for >1 month	88.2%
Chronic Diarrhoea for >1 month	68.1%
Persistent cough for >1 month	70.6%

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Based of sero-prevalence studies the DAC estimates to date more than 400,000 people have been infected HIV. The average sero prevalence rate in high risk behavior group like in multiple partner sexual contacts have reached to > 65% which was 17% in 1988 and sentinel sero-survey in representatives of general population example pregnant women attending clinics have shown a prevalence rate of 11-18% in 1992 in Addis Ababa and other regions. Among blood donors the sero prevalence have increased from 4.5% in 1989 to 7% 1992. In general it is estimated 5 - 10% of adults in urban towns have been infected with HIV.

Fever in AIDS

Since the training is mainly of Febrile illness few remarks on Fever and AIDS.

(1) Fever can come at early stage of HIV infection at the time of sero conversion. This is usually seen in 2 - 4 weeks after exposure to the virus and the clinical feature resembles that of any acute febrile illness. This acute clinical illness associated with primary HIV infection is said to occur in significant percent of ≥ 50%) HIV infected people.

(2) Fever is a major sign for AIDS diagnosis, out of total AIDS patients reported 88.20% fever was presenting sign in Ethiopia.

Persistent and recurrent fever common among HIV/AIDS infected patients in many cases the presumed cause is HIV itself and no other local infections that might explain the fever can be found.

How ever the following conditions also need to be considered in HIV/AIDS patients with fever

- Occult bacterial infection
  e.g pneumonia, sinusitis, UTI salmonella, RF.
- Mycobacterial infection e.g. M.tuber ulosis
- Fungal infection e.g. candidiasis
- Chronic virus infection e.g. CMV
- Parasitic infection e.g. malaria, to apologies
- Neoplasms, e.g. lymphones

Basic Facts about AIDS

HIV virus

The causative agent for AIDS is human immune deficiency virus which was isolated 1983 for first time. HIV is a member of lentevirus sub-family human retrovirus. Its main characteristics are long incubation period, and involvement of immune system and central nervus system HIV is fragile virus sensitive to even ultraviolet light and can be killed by high temperature and chemicals. Provided it is in conducive environment HIV can live in ambient Tem (7-15 days)

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Mode of Transmission and Prevention

Route of Exposure	Effectiveness	Contribution to Total	Prevention
Sexual	1%	75%	- Change of behavior - Abstaining - Careful selection of partner - Pre-testing before marriage - Condoms
Blood transfusion and using other blood producting	>90%	5%	- Limiting the need for transfusion - Screening of blood
Perinatal	20.40%	10%	Testing - Counseling Termination of pregnancy
IV drug use	0.5 - 1%	10%	- Change of - behavior - Avoid needle sharing - keep sterility
Occupational exposure needles type exposure	< 0.5%		- Universal precaution
Breast-feeding Injection and Scarification	Very low chance	-	

No evidence for transmission through

1. Saliva
2. Casual contact
3. Insect bite

What cells are infected with HIV?

1. T4-helper/Inducer Lymphocyte
2. Mononuclear cells
3. Macrophage, monocytes
4. Brain cells
5. Skin cells, Bowel Epitdium, Stem cells in Bone marrow etc.

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In which body fluid HIV present?

In High quantity	Moderate to low Quantity	Very Low Quantity
1. Blood, Plasma CSF	Genital secretion - Semen - Vaginal secretion	Breast milk Saliva, tear Urine, ear secretion Secretion - Sweats,

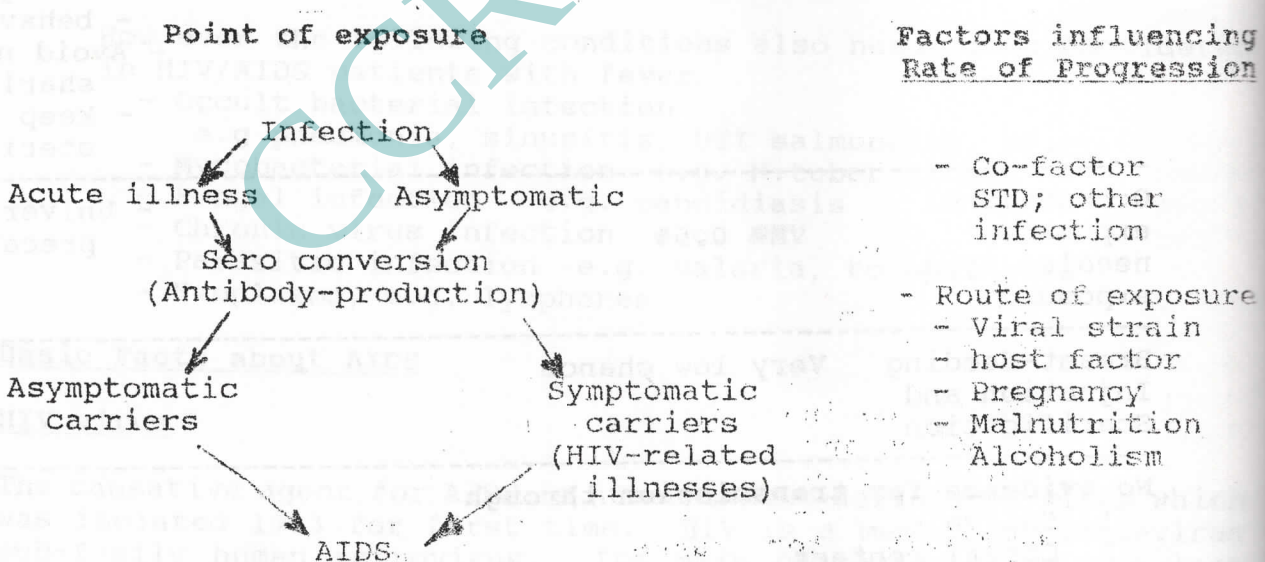
HIV infectivity depends?

1. Route
2. Frequency of exposure
3. Viral load, stages of the diseases
4. Immune status of the host
5. Host Genetic factors
6. Presence of co-factors e.g. STD
7. Viral strain

Natural history of HIV

AIDS is the end stage of HIV infection and the rate of progression varies greatly. It is said to be faster in developing countries compared to developed countries. In general it may take an average period of 5 to 10 years from infection to reach end stage AIDS.

Natural History of HIV Infection



- Predictors in HIV infected towards AIDS

. Low CD_4 count
. High antigen level
. Low Level of antibody
. Other Markers

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How to diagnose AIDS?

HIV infection does not mean AIDS various criterias developed to define AIDS depending on diagnostic capacity of each country i.e.

1. CDC AIDS case definition - USA and other developed countries
2. WHO Bangui AIDS case definition - For Africa
3. Caracas AIDS case definition - Latin America

Some countries have developed their own criteria for AIDS . In Ethiopia we have been using WHO Bangui with serology to define AIDS. The process of having Ethiopian AIDS case definition for surveillance purposes have been completed.

How does HIV causes disease?

1. Progressive destruction of immune system resulting immunosuppression.

2. Directly attacking tissues like
   Central nervous system
   GI - tracts
   Skin

How does it brings this changes?

Directly killing the infected cells, by creating Adhesion of infected cells (Syncytia).

- Disturbing the immune system regulation increased
  . i.e. Autoimmunity
  . Hypersensitivity

- By directly affecting stem cells in bone marrow

What happen then in AIDS?

- Destruction of immune system ----> increase risk of
  1. Infections
  2. Malignancy
- Direct attack of host organs
  eg. - Neurological impairment
  - Diarrhea
  - Skin problems

Common clinical features of HIV/AIDS patients

The symptoms and signs in HIV/AIDS patients can be as result of underlying opportunistic infection malignancy, or as result of direct effect of the virus on host organs. There is no single organ spared in AIDS. These days knowing AIDS is becoming as

ECRDA

knowing medicine. Although all organs and systems in the body can be affected by HIV/AIDS there are more common findings and specific diseases suggestive for HIV infection and AIDS.

Common symptoms and signs associated with HIV infection

1. Wight loss
2. Fever
3. Diarrhea
3. ulcers (persistent ulcers genital areas)
4. persistent cough
5. Neurological complaints/findings
6. Generalized Lymphadenopathy
7. Drug reactions (eg. thiacetazone, sulfonamide)
8. Skin infection - fungal, viral
9. Seborrhoeic dermatitis

Disease suggestive for HIV infection

1. Oral thrush
2. Tuberculosis
3. Kaposis sarcoma
4. Herpes zoster
5. Severe pruritus

Others very specific but difficult to diagnose in our local set up.

- Pneumocysts carina pneumonia
- Toxoplasmosis
- CMV
- Lymphomas
- Cryptoccocal meningite
- etc.

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Treatment

The care and support of HIV/AIDS patients depend on availability resource and capacity of the country which is the reflection of socio-economic status. In general treatment approaches include

1. Treatment of associated infections
2. Treatment of associated malignancies
3. Alleviating constitutional symptoms and signs
4. Antiviral agents and immune modulating agents
5. prevention of the occurrence of opportunistic infection by using prophylactic agents.

Majority of this options are not applicable and does not exist in most developing countries. In general the emphasis should be in alleviating symptoms and signs and treatment of treatable infections like Tuberculosis, candidiases and bacterial infections like pneumonia.

Occupational Risk and Prevention of HIV Transmission in Working place

HIV transmission can take place in health institutions from the patient to health care woke from one patient to another patient, or from health care work to the patient. Various studies have shown the risk exist but very minimal as compared to other blood born disease.

e.g. In hepatitis B. virus needle prick accident there is 20 times higher chance of acquire HSB as compared to HIV.

6. **Possible Routs in Health Institutions**

Exposure route	Effectiveness
- Needle Stick or (Sharp - ins. injury)	0.4 - 0.5%
- Mucosal exposure	?
- Open wound exposure	?

In one Study in U.S.A

out of 1514 who reported needle stick with AIDS patients - 4 cases infected (0.4%)

Majority of exposed HCW are nurses and physician
Nurse 60%
Physician 14%

Majority of this accidents occurred in pt. room or clinics accounting for 65% of the total exposure.

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- **Prevention of Transmission in Health Institution**

Universal Precautions

1. See all blood and body fluid as potentially infectious
2. The greatest risk is needle stick and puncture accident with sharp instruments so:
   - extra-ordinary care (concentration)
   - avoid re-capping of needles
   - avoid manipulating sharp instruments
3. Use disposable if possible if not - proper disinfection and sterilization before use.

4. During doing procedures when ever there is a risk of contact with body fluid or blood.

   a. wear gowns
   b. wear gloves
   c. wear Masks
   d. wash immediately after each procedure
   e. clean and disinfect the area
   f. disposed sharp instruments should be placed in puncture resistant containers.
   h. proper waste management (incineration)

5. Use proper sterile techniques and sterilization and disinfection methods.

   1. Steam sterilization (auto clavi)

      i.e Needles, syringe

      121°c for 2 hours

   2. Dry heat (dry ovum)
      eg. for metallic substances

      170°c for 2 hours

   3. Boiling - if others means are not available
      20 minutes.

   4. Chemical disinfection if other mens are not available
      immerse for 30 minutes.

      - glutaraldehyde

      - 70% ethanol
      - Hydrogen peroxide 6%

For decontamination of environmental surface

- Sodium hypochlorite is the most widely used chemical

WHO and CDC recently issued use bleach to cleat syringes and needles reduce the risk of HIV Transmission.

Wash with unused water	----->	bleach in the syringe 2-3 times for 30 seconds	-------->	Wash again Un used water

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