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Introduction

The first edition of the communicable diseases control guidelines, published in 1999, was widely appreciated and has been used as a reference for more than a decade. Given that Iraq is currently experiencing a period of growth, development, and reform, we feel it is an opportune time to write the second edition of these guidelines. This is an ongoing and evolving process – we hope to continue editing the guidelines every three years to provide medical and paramedical staff with the most up-to-date information as they work to promote public health, deal with outbreaks of communicable diseases, and improve early detection and surveillance systems through Iraq.

The guidelines cover the diseases that are currently included in the Iraq surveillance system. These diseases are broken down into three main groups:

- Group 1: Immediate Notification Diseases
- Group 2: Weekly Reported Diseases
- Group 3: Monthly Reported Diseases

Each individual guideline also includes:

1. Identification of the disease (Case Definition of Suspected, Probable, and Confirmed cases).
2. Infectious agent
3. Occurrence
4. Reservoir
5. Mode of Transmission
6. Incubation Period
7. Period of Communicability
8. Susceptibility and resistance
9. Methods of Control, which include:
   a. Preventive Measures
   b. Control of patient, contacts, and immediate environment
   c. Epidemic measures
   d. Disaster implications
   e. International measures
10. Management of the disease, including treatment, and case and contact management.
Group 1: Immediate Notification Diseases
1.1 Acute Flaccid Paralysis (AFP) Surveillance

ICD-10 AFP

It is an essential strategy of Polio Eradication Initiative (PEI), which aims to look for wild poliovirus circulation in the community, to investigate all possible polio cases. AFP surveillance is surveillance for all suspected or possible polio cases. Its purpose is to detect areas and groups where poliovirus transmission is occurring or likely to occur and to allow supplementary immunization to be focused where needed.

Case classification

*AFP case*: Any patient under 15 years of age with acute, flaccid paralysis, or any person of any age in whom a clinician suspects polio.

*Contact*: A contact is defined as a child less than 5 years of age who has been in direct contact with the index AFP cases within one week prior to the onset of paralysis and/or within two weeks after onset of paralysis.

Types of AFP surveillance

1. **Routine surveillance for AFP (“zero reporting”)**
   Immediate notification of AFP in children <15 years of age is required. AFP should also be included in the weekly and monthly reporting system. When no case of AFP is detected, reporting units should still send weekly and monthly reports indicating zero cases.

2. **Active surveillance for AFP**
   Active surveillance is a strategy to actively collect information by visiting health facilities. A designated person should make visits to sites likely to have cases of acute polio, such as hospitals and rehabilitation centers. An active surveillance focuses mainly on hospitals, because most children with sudden paralysis will be admitted to hospitals or end up in hospitals because of referral.

3. **Active AFP case finding**
   Looking for AFP cases in the community.

Key components to Acute Flaccid Paralysis surveillance

- Detect and investigate all cases of AFP in children <15 years.
- Collect 2 stool specimens, collected at least 24 hours apart, within 14 days of onset of paralysis.
- Conduct virological testing of stool specimen in *WHO*-accredited lab.
- Conduct 60 day follow-up for residual paralysis.
- Classify cases according to WHO scheme.
Key indicators of surveillance performance

- Non-polio AFP rate (Target: 2/100000 population <15 years annually)
- 2 stool specimens collected more than 24 hours apart and within 14 days from date of onset of paralysis. Each specimen of adequate volume (8-10g), packed and shipped in a cold box, and arriving within 3 days of collection to the lab in good condition (Target: 80%)

Management of this disease

- One in 200 infections leads to irreversible paralysis, usually in the legs. This is caused by the virus entering the blood stream and invading the central nervous system. As it multiplies, the virus destroys the nerve cells that activate muscles. The affected muscles are no longer functional and the limb becomes floppy and lifeless – a condition known as acute flaccid paralysis (AFP). All cases of acute flaccid paralysis (AFP) among children under fifteen years of age are reported and tested for poliovirus within 48 hours of onset.

- Around 40% of people who survive paralytic polio may develop additional symptoms 15–40 years after the original illness. These symptoms – called post-polio syndrome – include new progressive muscle weakness, severe fatigue and pain in the muscles and joints.

- There is no cure for polio, only treatment to alleviate the symptoms. Heat and physical therapy is used to stimulate the muscles and antispasmodic drugs are given to relax the muscles. While this can improve mobility, it cannot reverse permanent polio paralysis.

- Polio can be prevented through immunization. Polio vaccine, given multiple times, almost always protects a child for life.

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1.2 Anthrax

ICD-10 A22

1.2.1 Identification

Anthrax is an acute bacterial disease usually affecting the skin, and rarely involving the respiratory or intestinal tracts. Most forms of the disease are lethal. Anthrax affects both humans and other animals. Anthrax has an acute onset characterized by several clinical forms. These include:

1. Localized form (cutaneous): skin lesion usually on an exposed part of the body such as the face, the neck or arm, evolving over 7 days from a papular through a vesicular stage, to a depressed black eschar invariably accompanied by edema that may be mild to extensive.

2. Systemic forms
   2.1. pulmonary (inhalation), characterized by brief prodrome resembling acute viral respiratory illness, followed by rapid onset of hypoxia, dyspnea, and high temperature, with Chest X-ray evidence of mediastinal widening.
   2.2. Gastro-intestinal: characterized by abdominal distress, with nausea, vomiting, anorexia and followed by fever.
   2.3. Meningeal: acute onset of high fever possibly with convulsions, loss of consciousness, meningeal signs and symptoms, commonly noted in all systemic infection.

Case classification:

1. Suspected case: A case that is compatible with the clinical description and has an epidemiological link to confirmed or suspected animal cases or contaminated animal product.

2. Probable case: A suspected case that has a positive reaction to allergic skin test (in non-vaccinated individuals)

3. Confirmed case: Positive serology (PCR, IFAT, ELISA)

Laboratory criteria for diagnosis:

1. Isolation of *Bacillus anthracis* from a clinical specimen (blood, lesions, discharges).
2. Demonstration of *B. anthracis* in a clinical specimen by microscopic examination of stained smears (vesicular fluid, blood, cerebrospinal fluid, pleural fluid, stools).
3. Positive serology (ELISA, Western blot, toxin detection, chromatographic assay, fluorescent antibody test (FAT).

Note: It may not be possible to demonstrate *B. anthracis* in clinical specimen if the patient has been treated with antimicrobial agents.

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1.2.2 Infectious agent

*Bacillus anthracis*, a gram-positive, encapsulated, spore forming, non-motile aerobic rode.

1.2.3 Occurrence

Cases of anthrax, 2000-2009

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</tr>
</tbody>
</table>

Agricultural anthrax occurs especially among veterinarians, agricultural workers and butchers, and industrial anthrax, resulting from exposure to contaminated sheep wool or goat hair that are processed into yarns used in the textile and carpet industry, as well as cattle hides that are processed into leather goods, or bones used for the manufacture of gelatin and/or fertilizer.

1.2.4 Reservoir

Dried or otherwise processed skins and hides of infected animals may harbor spores for years. Spores also remain viable in contaminated soil for many years.

1.2.5 Mode of transmission

1. Through skin lesions by contact with tissues of infected animals (cattle, sheep, goats, etc.)
2. Gastrointestinal anthrax: through ingestion of contaminated food (uncooked meat) or inhalation of spore-laden dust.

1.2.6 Incubation period

Most cases occur within 2-7 days of exposure; however, an incubation period of up to 60 days is possible.

1.2.7 Period of communicability

There is no evidence of direct spread from person to person. Articles and soil contaminated with spores may remain infective for years.

1.2.8 Susceptibility and resistance

The case fatality rate of cutaneous anthrax usually is about 20% if untreated. Systemic infection resulting from inhalation causes a case fatality rate of 100% and gastrointestinal causes death in 25% to 75% of cases, if untreated. Recovery is usually followed by prolonged immunity.
1.2.9 Methods of control

1.2.9a Preventive measures:
- Educate employees who are handlers of potentially infected articles in the proper care of skin abrasions.
- Ensure proper ventilation in hazardous industries and use of protective clothing.
- Sterilize hair, wool or hides, bone meal or other feed of animal origin prior to processing.
- Use vaporized formaldehyde for terminal disinfection of textile mills contaminated with *B. anthracis*.
- Deeply bury carcasses with quicklime at site of death, if possible. Do not necropsy or burn on open field.
- Decontaminate soil or discharges with quicklime, or preferably bury deeply with carcass.
- Vaccinate all animal at risk, and revaccinate annually.

1.2.9b Control measures
- Treatment of the patient (penicillin is drug of choice, tetracycline, erythromycin, chloramphenicol, ciprofloxacin could be used).
- Arrange isolation of patient in hospital, soiled articles require pressure steam sterilization or incineration.
- Contacts tracing.

1.2.10 Management of the disease

1.2.10a Mild uncomplicated cases
- In mild uncomplicated cases of cutaneous anthrax, penicillin V, 500 mg, taken orally every 6 hours for 5-7 days is adequate, but the treatment usually recommended is 3 to 7 days of intramuscular procaine penicillin, 600 mg (1 million units), every 12.24 hours or intramuscular benzyl penicillin (penicillin G), 250 000 units at 6 hourly intervals.
- Cutaneous lesions usually become sterile within the first 24 hours of such regimens but, although early treatment will limit the size of the lesion, it will not alter the evolutionary stages it must go through.

1.2.10b Severe or potentially life-threatening cases
- In severely affected patients or when pulmonary or gastrointestinal anthrax is suspected, the initial treatment is penicillin G, 1200 mg (2 million units) per day by infusion or by slow intravenous injection (<300 mg/min) every 4-6 hours until the patient’s temperature returns to normal; at this point treatment should continue in the form of intramuscular procaine penicillin administered as described above.
- Streptomycin, 1.2 g per day intramuscularly, may act synergistically with penicillin.
- General measures for treatment of shock may be life saving since death is due, at least in part, to toxin-induced shock. Intubation, tracheotomy or ventilatory support may

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be required in the event of respiratory problems, and vasomotor support with dopamine may be necessary when there is hemodynamic instability.

- Primary hematological, renal or liver function disorders are not generally seen.

1.2.10c Alternatives to penicillin

- In the event of allergy to penicillin, several antibiotics are effective alternatives, including tetracyclines, chloramphenicol, gentamicin and erythromycin. Of the tetracycline family, tests in animals have indicated doxycycline is very effective and that the quinolone, ciprofloxacin may also be suitable.

- Trimethoprim is not effective.

Note: Coordination between Agriculture and Health Ministries is essential.

http://www.who.int/vaccine_research/diseases/zoonotic/en/index1.html

A skin lesion caused by anthrax

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4 CDC/ James H. Steele
1.3 Cholera

ICD-10 A00

1.3.1 Identification

Cholera is an acute bacterial enteric disease characterized in its severe form with sudden onset, profuse painless watery stools (rice-water stool), nausea and profuse vomiting. Spasmodic abdominal pain might occur. Severe muscle cramps in extremities may be due to electrolyte disturbance. In untreated cases, rapid dehydration, acidosis, circulatory collapse, hypoglycemia in children and renal failure may occur. Death results within a few hours in 50% of severe cases, however, if proper treatment is started early, mortality may reach only 1%.

Cholera is essentially the only diarrheal disease where patients can become severely dehydrated in less than six hours. No one who arrives at a treatment center and is still breathing should die of cholera.

Case classification
1. **Suspected case**: Any case complains of acute watery diarrhea without pain, +/- vomiting, regardless of patient age.
2. **Probable case**: Any suspected case with severe dehydration, or death due to acute watery diarrhea.
3. **Confirmed case**: A suspected case with isolation of *Vibrio cholera* O1 or O139 from stools.
4. **Carrier**: Asymptomatic person *Vibrio cholera* isolated from his or her stool

1.3.2 Infectious agent

*Vibrio cholerae* serogroup O1 and O139. Serogroup O1 occurs as two biotypes- classical and El Tor- each of which occurs as 3 serotypes (Inaba, Ogawa and rarely Hikojima). A serogroup is defined by its agglutination with a specific antiserum. If the strain does not agglutinate with the antisera; it is known as a non-O1, non-O139 *vibrio cholera* (NAG). The cholera toxin is primarily responsible for fluid loss.

1.3.3 Occurrence

Cholera is one of the oldest and best-understood epidemic diseases. Epidemics and pandemics are strongly linked to the consumption of unsafe water and food, poor hygiene, poor sanitation and crowded living conditions.

1.3.4 Reservoir

Humans only.
1.3.5 Mode of transmission

Consumption of food or water contaminated directly or indirectly with stool or vomitus of a case (e.g. stored unsafe water in highly populous areas because bacteria can stay in water for a long time).

1.3.6 Incubation period

From few hours to 5 days. Range 1-3 days.

1.3.7 Period of communicability

As long as stools are positive, usually only a few days after recovery.

1.3.8 Susceptibility and resistance

Variable; gastric achlorhydria increases the risk of illness, and breastfed infants are protected. Severe cholera occurs significantly more often among persons with blood group O. Infection with either *V.cholera* O1 or O139 results in a rise in agglutinating and antitoxic antibodies, and increased resistance to re-infection.

1.3.9 Methods of control

1.3.9a Preventive measures

- Simple improvement in water, hygiene, and sanitation can significantly reduce the incidence of cholera and other diarrheal diseases.
- Wash hands with soap after using toilets and latrines, before preparing food, and before eating.
- Boil water or disinfect water with chlorine solution.
- Only eat freshly cooked food.
- Do not defecate near water sources.
- Use latrines and keep them clean.
- Encourage breastfeeding throughout infancy; boil all milk and water used for infant feeding.

1.3.9b Control measures

All cases should be reported by health care providers.

Surveillance procedures:

- Reporting: patients with suspected or confirmed cholera should be reported immediately to DSU, GSU, and CSU.
- Case investigation: all suspected cases should be immediately investigated by DSU in collaboration with the Communicable Disease Program. Case investigation forms should be completed and immediately sent to the Communicable Disease Program...
and CSU. It is important to coordinate surveillance efforts with the Central Public
Health Laboratory to ensure appropriate collection and handling of laboratory
specimens.

**Laboratory procedure:** Fresh stool samples taken in Cary-Blair media and cultures in
alkaline peptone water should be sent at room temperature to common labs for a maximum
of 6 hours.

**Data analysis:** Routine weekly and monthly reporting of aggregate data is recommended
from peripheral level to intermediate and central levels. Immediate notification for every case
to CSU is required. Zero reporting is mandatory when there is no case.

**Actions to be taken:**
- The most important action for managing patients with cholera and other severe
dehydrating diarrheal diseases is rapid and appropriate rehydration to make up for the
fluid losses that have occurred prior to coming for treatment, and maintenance
hydration to compensate for the ongoing fluid losses. This is done by either ORS or
intravenous fluids according to dehydration status.
- **For cases:** Oral rehydration therapy is the main stay of therapy for patients with acute
watery diarrhea. Antimicrobial therapy is an important adjunct to oral rehydration
therapy of patient with cholera.
- **For contacts:** Surveillance for acute disease should be conducted among contacts
who shared food or water with cholera patients. In settings where household
transmission is likely, contacts should be treated with prophylactic antibiotics.

1.3.9c Epidemic measures

Health education and improving sanitation are effective containment measures.

1.3.9d Disaster implications

Outbreak risks are high in endemic areas if large groups of people are crowded together
without a sufficient quantity of safe water, adequate food handling, or sanitary facilities.

1.3.9e International measures

1. Governments are required to report cholera cases due to *V.cholera* O1 and O139, and
outbreaks or epidemics of acute watery diarrhea, to WHO when they are unusual or
unexpected, or when they present significant risk of international spread or of
international travel or trade restrictions (International Health Regulations 2005).
2. International regulations states that information should include number of new cases,
number of deaths, age distribution, and hospital admissions.
3. Measures applicable to ships, aircraft and land transport arriving from cholera areas
are to be applied within the framework of the revised International Health
Regulations 2005.
4. International travelers: cholera vaccination proof is not required as a condition for
entry by any country.
1.3.10 Management of the disease

- Efficient treatment resides in prompt rehydration through the administration of oral rehydration salts (ORS) or intravenous fluids, depending on the severity of cases. Up to 80% of patients can be treated adequately through the administration of ORS (WHO/UNICEF ORS standard sachet).
- Very severely dehydrated patients are treated through the administration of intravenous fluids, preferably Ringer lactate. Appropriate antibiotics can be given to severe cases to diminish the duration of diarrhea, reduce the volume of rehydration fluids needed and shorten the duration of *V. cholerae* excretion.
- For children up to five years, supplementary administration of zinc\(^2\) has a proven effective in reducing duration of diarrhea as well as reduction in successive diarrhea episodes. In order to ensure timely access to treatment, cholera treatment centers should be set up among the affected populations whenever feasible.

---


\(^6\) [http://phil.cdc.gov](http://phil.cdc.gov)
1.4 Crimean-Congo Hemorrhagic Fever

ICD-10 A98

1.4.1 Identification

A viral disease with sudden onset of fever, malaise, weakness, irritability, headache, pain in limbs and bleeding tendency manifested at fourth day of illness, which may include epistaxis, bleeding gum, G.I.T or urogenital tract bleeding, echymosis and purpura (spots with bruising patches on skin), and bleeding from the site of injection.

Case definitions

Suspected case: Fever with acute hemorrhagic symptoms with history of contact with animal.
Probable case: Suspected case plus history of contact with confirmed human case.
Confirmed case: Suspected case with laboratory confirmatory tests. Diagnosis confirmed by C.F.T, IFAT, ELISA.

1.4.2 Infectious agent

The Crimean-Congo hemorrhagic fever virus (*Bunyaviridae Nairovirus*)

1.4.3 Occurrence

Situation analysis in Iraq of HF, 2000 – 2011

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<td>3</td>
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<td>2</td>
<td>10</td>
<td>6</td>
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</table>

In 2010, an outbreak in Ninawa province occurred. No. of confirmed cases were 10 and one case occurred in Duhok province. 3 of these cases died. Death rate is 10 – 30% in Iraq.

1.4.4 Reservoir

Tick (*Hyalomma marginatum*) are both a reservoir and a vector, domestic animals (sheep, goats and cattle) may act as amplifying transmission host during epizootics.

1.4.5 Mode of transmission

1. By bite of infective *Hyalomma marginatum*.
2. Nosocomial infection.
3. Butchering infected animal: spread by infected fluid especially blood through direct contact with injured or scratch human skin.
1.4.6 Incubation period

3-12 days. Depends on the mode of acquisition of the virus. With tick bite: 1 to 3 days, up to 9 days. Following contact with infected blood or tissues: 5 to 6 days, up to 12 days.

1.4.7 Period of communicability

Domestic animals, such as cattle, sheep and goats, are viremic (virus circulating in the bloodstream) for around one week after becoming infected with ticks.

1.4.8 Susceptibility and resistance

Recovery is slow, begins on the ninth or tenth day after the onset of illness. Case fatality rates in hospitalized patients have ranged from 9% to as high as 50%.

1.4.9 Methods of control

1. Case detection and report to Health Department in the province within 24 hours by telephone or E-mail and also to CDC.
2. Isolate suspected human cases and confirmed cases in the hospitals with control measures to protect medical and paramedical contacts.
3. Tics control measures
4. Check for the close contacts in patient’s house under medical supervision by nearby PHC. Contacts with tissue or blood exposure from patients with suspected or confirmed cases should be followed up with daily temperature and symptom monitoring for at least 14 days after the exposure.
5. Rapid information to veterinary department to take action by using tick insecticides on the infested animal at the patient’s house.

1.4.10 Management of the disease

- General supportive therapy is the mainstay of patient management in CCHF. Intensive monitoring to guide volume and blood component replacement is required.
- The antiviral drug ribavirin has been used in treatment of established CCHF infection with apparent benefit. Both oral and intravenous formulations seem to be effective.
- The value of immune plasma from recovered patients for therapeutic purposes has not been demonstrated, although it has been employed on several occasions.

---


Isolated male patient diagnosed with Crimean-Congo hemorrhagic fever

9

Centers for Disease Control and Prevention's Public Health Image Library (PHIL)
1.5 Diphtheria

ICD-10 A36

1.5.1 Identification

Upper respiratory tract illness with laryngitis or pharyngitis or tonsillitis, plus adherent membranes on tonsils or nasopharynx.

**Case classification**

1. **Suspected case**: not applicable.
2. **Probable case**: Case that meets the clinical description
3. **Confirmed case**: Probable case confirmed by laboratory (isolation of *C. diphtheriae* from a clinical specimen), or epidemiologically linked to a laboratory-confirmed case.
4. **Carrier**: presence of *C. diphtheriae* in nasopharynx but no symptoms. **NOTE**: persons positive with *C. diphtheriae* identification but who do not meet the clinical descriptions (asymptomatic carriers) should NOT be reported as probable or confirmed cases.

1.5.2 Infectious agent

Bacterium *Corynebacterium diphtheriae*

1.5.3 Occurrence

Diphtheria is endemic in Iraq\(^\text{10}\).  

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1.5.4 Reservoir

Humans are the only known reservoirs.

1.5.5 Mode of transmission

- Contact (usually direct, rarely indirect) with the respiratory droplets of a case or carrier
- In rare cases, the disease may be transmitted through foodstuffs (raw milk has served as a vehicle)

1.5.6 Incubation period

Usually 2-5 days, occasionally longer.

1.5.7 Period of communicability

---

Untreated patients are infectious for 2 to 3 weeks - until virulent bacilli have disappeared from discharges and lesions. The rare chronic carrier can shed bacilli for 6 months or more. The disease is usually not contagious 48 hours after antibiotics are instituted.

1.5.8 Susceptibility and resistance

People most susceptible to infection are those who are not completely immunized and have been exposed to a carrier or diseased individual.

1.5.9 Methods of control

The control of diphtheria is based on 3 measures:
1. Ensuring high population immunity through vaccination (primary prevention).
3. Early diagnosis and proper case management (tertiary prevention of complications and deaths).

1.5.9a Preventive measures (Immunization)

- Doses of 0.5 ml DTP intramuscularly in outer part of thigh, according to national schedule
- If immunization is started later, there must still be an interval of 4 weeks between doses.
- Immunization to be completed preferably before the age of 6 months (26 weeks).
- DTP vaccine must be stored between +2°C and +8°C.
- DTP vaccine can be given to immunocompromised children up to 7 years old; Td vaccine can be given to immunocompromised adults.

1.5.9b Control measures (Case management)

Diphtheria antitoxin and antibiotic therapy are the cornerstone of therapy for diphtheria. The antibodies only neutralize toxin before its entry into cells, and is therefore critical that diphtheria antitoxin be administered as soon as a presumptive diagnosis has been made. Antibiotic therapy, by killing the organism, has three benefits:
- The termination of toxin production
- Amelioration of the local infection
- Prevention of spread of the organism to uninfected persons

*Do not wait for laboratory results before starting treatment / control activities.*

**Patients**
Diphtheria antitoxin IM (20,000 to 100,000 units) in a single dose, immediately after throat swabs have been taken. Procaine penicillin G IM (25,000 to 50,000 units/kg/day for children; 1.2 million units/day for adults in 2 divided doses) or parenteral erythromycin (40-50 mg/kg/day with a maximum of 2 g/d) until the patient can swallow; *then* Oral penicillin V (125-250 mg) in 4 doses a day, or oral erythromycin (40-50 mg/kg/day with a maximum of 2 g/d) in 4 divided doses. *Antibiotic treatment should be continued for a total period of 14 days.* Isolation: strict (pharyngeal diphtheria) or contact (cutaneous diphtheria) for 14 days.
Communicable Disease Control Guidelines

Note: Clinical diphtheria does not necessarily confer natural immunity, and patients should therefore be vaccinated before discharge from a health facility.

Close Contacts
Surveillance for 7 days for all persons with close contact, regardless of vaccination status, and throat culture. All must receive a single dose of benzathine penicillin G IM (600,000 units for children < 6 years; 1.2 million units for 6 years or older). If culture is positive, give antibiotics as for patients above.

Carriers
All must receive a single dose of benzathine penicillin G IM (600,000 units for children < 6 years; 1.2 million units for 6 years or older).

1.5.9c Epidemic measures

- Inform the Health Authorities if one or more suspected cases are identified.
- Confirm the suspected outbreak, following WHO guidelines.
- Investigate any probable case: check if it fulfills the case definition, record date of onset, age and vaccination status.
- Confirm the diagnosis: collect both nasal and pharyngeal swabs for culture and swabs from any wounds or skin lesions. If appropriate facilities are available, determine the biotype and toxigenicity of \textit{C. diphtheriae}.
- Identify close contacts and define population groups at high risk. Adult contacts must avoid contact with children and must not be allowed to undertake food handling until proven not to be carriers.
- Implement outbreak response measures. Give priority to case management and immunization of population in areas not yet affected where the outbreak is likely to spread.
- Immunize the population at risk as soon as possible, especially children. In an epidemic involving adults, immunize groups that are most affected and at highest risk. Repeat immunization procedures 1 month later to provide at least 2 doses to recipients.
- In epidemic situations, preferably Td vaccine (a combination of diphtheria and tetanus toxoids with reduced diphtheria content) should be given.
- To ensure safety of injection during immunization, auto disable syringes and safety boxes are recommended. Safe disposal of used sharps should be ensured.

1.5.10 Management of the disease\textsuperscript{11}

1.5.10a Case Management

- If diphtheria is strongly suspected, specific treatment with antitoxin and antibiotics should be initiated immediately.
- \textit{Do not wait for laboratory results before initiating treatment.}

\textsuperscript{11} WHO. “Communicable Disease Toolkit Iraq Crisis: Case Management of Epidemic-Prone Diseases” March 2003. \url{http://www.who.int/infectious-disease-news/IDdocs/whocds200317/7casemg.pdf}
- I.M. antitoxin is the mainstay of treatment: 20 000 to 100 000 units in a single dose, immediately after throat swabs have been taken.
- Antibiotics are necessary to eliminate the organism and prevent spread; they are not a substitute for antitoxin treatment.
- Recommended dose regimens are:
  - procaine penicillin IM (25 000 to 50 000 units/kg/day for children; 600 000 units/kg/day for adults in 2 divided doses) or parenteral erythromycin (40-50 mg/kg/day) with a maximum of 2 g/d until the patient can swallow
    then
  - oral penicillin V (125-250 mg) in 4 doses a day or erythromycin (40-50 mg/kg/day in 4 divided doses)
- Antibiotic treatment should be continued for 14 days
- Note: Clinical diphtheria does not necessarily confer natural immunity, and patients should therefore be vaccinated before discharge from a health facility

1.5.10b Management of close contacts
- Close contacts include household members and other persons with a history of direct contact with a case, as well as health care staff exposed to oral or respiratory secretions of a case
- All should be clinically assessed for symptoms and signs of diphtheria and kept under daily surveillance for 7 days from last contact with the case
- Adult contacts must avoid contact with children and must not be allowed to undertake food handling until proven not to be carriers
- All must receive a single dose of benzathine penicillin G IM (600 000 units for children)
1.6 Influenza H1N1, H5N1

ICD-10 J09: Avian
ICD-10 J10: Seasonal
ICD-10 J11: Virus Not Identified

1.6.1 Identification

Viral infection that affects mainly the nose, throat, bronchi and, occasionally, lungs. Although uncomplicated influenza-like illness (fever, cough or sore throat) has been reported in many cases, mild respiratory illness (nasal congestion, rhinorrhea) without fever and occasional severe disease also has been reported. Other symptoms reported with swine influenza A virus (H1N1) infection include vomiting, diarrhea, myalgia, headache, chills, fatigue, and dyspnea. Conjunctivitis is rare, but has been reported. Severe disease (pneumonia, respiratory failure) and fatal outcomes have been reported with swine influenza A virus infection.

Case classification

Confirmed case: of influenza A (H1N1) virus infection: a person with an acute respiratory illness with laboratory confirmed influenza A (H1N1) virus infection at CDC by viral culture or four-fold rise in swine influenza A (H1N1) virus-specific neutralizing antibodies.

Suspected case: of influenza A (H1N1) virus infection: a person with acute febrile respiratory illness with onset within 7 days of close contact with a person who is a confirmed case of influenza A (H1N1) virus infection.

Close contact: a person within about 6 feet of an ill person who is a confirmed or suspected case of influenza A (H1N1) virus infection.

1.6.2 Infection agent

Influenza viruses: Type A, B and C. Most common cause of annual outbreaks and epidemics are Type A and B.

Influenza A virus subtypes reported in severe epidemics includes avian H5N1, H7, and swine H1N1 and H3N2 viruses.

1.6.3 Occurrence

Annual outbreaks of influenza occur most commonly during autumn and winter months.

Influenza A can cause severe epidemics (as well as severe worldwide epidemics; or pandemics) among all ages. Influenza B viruses in general are associated with less severe epidemics (chiefly among children) than influenza A viruses, they have not caused pandemics. Influenza C virus causes only mild disease and has not been associated with widespread epidemics or pandemics.
1.6.4 Reservoir

Influenza type A infects multiple species including people, birds, pigs, horses, and other animals. Wild birds are the natural hosts for these viruses. All birds are thought to be susceptible to infection with avian influenza, though some species are more resistant to infection than others. Infection causes a wide spectrum of symptoms in birds, ranging from mild illness to a highly contagious and rapidly fatal disease resulting in severe epidemics.

Influenza B viruses are usually found only in humans.

1.6.5 Mode of transmission

Seasonal Influenza or pandemic H1N1 is transmitted from person to person via droplets and aerosols produced when infected people cough or sneeze.

Avian influenza A (H5N1) virus transmission occur through bird-to-human: Direct contact with infected poultry, or with surfaces and objects contaminated by their droppings, is the main route of transmission to humans.

1.6.6 Incubation period

About 2 days

1.6.7 Period of communicability

Persons with pandemic influenza A (H1N1) virus infection should be considered potentially contagious for up to 7 days following illness onset. Persons who continue to be ill longer than 7 days after illness onset should be considered potentially contagious until symptoms have resolved. Children, especially younger children, may be contagious for longer periods.

Non-hospitalized ill persons or are confirmed or suspected cases of swine influenza A (H1N1) virus infection are recommended to stay at home (voluntary isolation) for at least the first 7 days after illness onset except to seek medical care.

1.6.8 Susceptibility and resistance

Annual outbreaks of influenza are due to minor changes in the surface proteins of the viruses that enable the viruses to evade the immunity humans have developed after previous infections with the viruses or in response to vaccinations. When a major change in either one or both of their surface proteins occurs spontaneously, no one will have partial or full immunity against infection because it is a completely new virus. If this new virus also has the capacity to spread from person-to-person, then a pandemic is most likely to occur.

While most people recover from a bout of influenza, there are large numbers of people who need hospital treatment and some may die due to complications especially children younger than age two, adults age 65 or older, and people of any age with chronic heart and respiratory illnesses.
1.6.9 Methods of control

1.6.9a Preventive measures

- People should cover their mouth and nose with a tissue when coughing, and wash their hands regularly.
- The most effective way to prevent the disease or severe outcomes from the illness is vaccination. Vaccination should be considered for high-risk groups.

1.6.10 Management of the disease\textsuperscript{12}

- Mild illness continues to characterize most cases, and basic supportive care (to relieve aches or fever) is sufficient for most people. However, health care providers should give all of their patients guidance on how to recognize signs of progressive illness, and when to seek medical attention.
- For pregnant women, WHO advises early antiviral treatment for suspected or confirmed pandemic influenza illness.
- Infants and very young children (those under 2 years of age), especially those with underlying conditions, should also be treated with antiviral medication if warning symptoms arise.
- In general, antiviral treatment recommendations are:
  - Patients who have severe or progressive illness should be treated with antiviral medication as soon as possible.
  - People with mild symptoms but who are at higher risk for severe illness (e.g. pregnant women, infants and young children, and those with chronic lung problems) should start antiviral treatment as soon as possible.
  - Antiviral treatment is not necessary for people have uncomplicated, or mild, illness and are not in a high risk group for severe illness.
  - Mothers who are breastfeeding can continue breastfeeding while ill and receiving antiviral treatment.
- In hospital settings, health providers should monitor oxygen levels closely and supplement oxygen as needed, following guidelines. When pneumonia is present patients should be treated with both antiviral medication and antibiotics as early as possible.

1.7 Plague

ICD-10 A20

1.7.1 Identification

A specific zoonosis disease characteristic with rapid onset of fever, chills headache, severe malaise prostration. The disease can occur in 3 forms: 1) Bubonic form: extreme painful swelling of lymph glands (buboes); 2) Pneumonic form: cough with blood-stained sputum, chest pain, and difficult breathing; and 3) Sepsis: circulatory collapse, coagulopathy, hemorrhage, respiratory distress, shock, and organ failure, leading to death.

The disease involves rodents (reservoir) and their fleas (vector) which transfer the bacterial infection to various animals and to people.

Laboratory diagnosis: microscopic finding of the gram-negative bipolar coccobacilli in clinical specimen (bubo aspirate, sputum, tissue or blood) or antigen or antibody detection in clinical specimens using IFAT, ELISA.

Note: It is subjected to international health regulation.

1.7.2 Infectious agent

*Yersinia pestis*, the plague bacillus.

1.7.3 Occurrence

There have been no reported cases in Iraq.

1.7.4 Reservoir

- Wild rodents are the natural vertebrate reservoir of plague.
- Rabbits, hares and domestic cats may also be a source of infection.

1.7.5 Mode of transmission

- Transmitted from rodent to human by the bite of an infected flea vector.
- Human-to-human transmission comes from inhalation of infected droplets spread by coughing patients with plague pneumonia or pharyngitis or by direct contact with pus from suppurating buboes.

1.7.6 Incubation period

From 1-7 days; may be a few days longer in immunized individuals. For primary plague pneumonia, usually shorter.
1.7.7 Period of communicability

Fleas may remain infective for months under suitable conditions of temperature and humidity. The most communicable form is pneumonic plague; overcrowding facilitates transmission. Patients with pneumonic plague are infectious until completion of at least 48 hours of appropriate antibiotic therapy. Patients with draining buboes are communicable until lesions are surgically excised or heal.

1.7.8 Susceptibility and resistance

*Y. pestis* is an extremely virulent organism mostly resulting from its virulence factors that impair the host innate immunity response. The case fatality rate is close to 50-100% if no antibiotic treatment is given within the first 48 hours following symptoms onset.

1.7.9 Methods of control

1. Report to local health authority, CDC urgently by phone or e-mail.
2. Use a safe insecticide to rid the patient (including clothing and baggage) of fleas.
3. Hospitalize & isolate the case.
4. Concurrent disinfection of sputum, purulent discharges and soiled articles.
5. Terminal clean.
6. Handle bodies of plague victims with strict aseptic precautions.
7. Streptomycin is the drug of choice, gentamycin could be used when streptomycin is not available; tetracyclines and chloramphenicol are alternative choices.
8. Investigation of the contacts and search for the source of infection.

1.7.10 Management of the disease

- When a diagnosis of human plague is suspected on clinical and epidemiological grounds, appropriate specimens for diagnosis should be obtained immediately and the patient should be started on specific antimicrobial therapy without waiting for a definitive answer from the laboratory.
- Suspect plague patients with evidence of pneumonia should be placed in isolation, and managed under respiratory droplet precautions.
- **Specific therapy**
  - *Aminoglycosides: streptomycin and gentamicin*: Streptomycin is the most effective antibiotic against *Y. pestis* and the drug of choice for treatment of plague, particularly the pneumonic form. Therapeutic effect may be expected with 30 mg/kg/day (up to a total of 2 g/day) in divided doses given intramuscularly, to be continued for a full course of 10 days of therapy or until 3

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days after the temperature has returned to normal. Gentamicin has been found to be effective in animal studies, and is used to treat human plague patients.

- **Supportive therapy**
  - The clinician must prepare for intense supportive management of plague complications, utilizing the latest developments for dealing with Gram-negative sepsis. Aggressive monitoring and management of possible septic shock, multiple organ failure, adult respiratory distress syndrome (ARDS) and disseminated intravascular coagulopathy should be instituted.

- **Treatment of plague during pregnancy and in children**
  - With correct and early therapy, complications of plague in pregnancy can be prevented. The choice of antibiotics during pregnancy is confounded by the potential adverse effects of three of the most effective drugs. Streptomycin may be ototoxic and nephrotoxic to the fetus. Tetracycline has an adverse effect on developing teeth and bones of the fetus. Chloramphenicol carries a low risk of "grey baby" syndrome or bone-marrow suppression. Experience has shown that an aminoglycoside judiciously administered is effective and safe for both mother and fetus, and in children. Because of its safety, intravenous or intramuscular administration, and ability to have blood concentrations monitored, gentamicin is the preferred antibiotic for treating plague in pregnancy.

- **Prophylactic therapy**
  - Persons in close contact with pneumonic plague patients, or persons likely to have been exposed to *Y. pestis*-infected fleas, to have had direct contact with body fluids or tissues of a *Y. pestis*-infected mammal, or exposed during a laboratory accident to known infectious materials should receive antibiotic preventive therapy, if the exposure was in the previous six days. The preferred antimicrobials for preventive or abortive therapy are the tetracyclines, chloramphenicol, or one of the effective sulfonamides.

- **Hospital precautions**
  - Standard patient-care precautions should be applied to management of all suspected plague patients. These include prescribed procedures for hand washing, wearing of latex gloves, gowns, and protective devices to protect mucous membranes of the eye, nose and mouth during those procedures and patient-care activities likely to generate splashes or sprays of blood, body fluids, secretions and excretions. Additionally, a patient with suspected respiratory plague infection should be specifically managed under respiratory droplet precautions, including management in an individual room, restriction of movement of the patient outside the room, and masking of the patient as well as persons caring for the patient until the patient is no longer infectious.

http://www.who.int/vaccine_research/diseases/zoonotic/en/index1.html
Acral necrosis of the nose, the lips, and the fingers and residual ecchymoses over both forearms in a patient recovering from bubonic plague that disseminated to the blood and the lungs.

1.8 Pertussis (whooping cough)

ICD-10 A37

1.8.1 Identification

A highly contagious upper respiratory tract bacterial infection. Clinically has 3 stages; the initial catarhal stage is characterized by the insidious onset of coryza (runny nose), sneezing, low-grade fever, and a mild, occasional cough, similar to the common cold. The cough gradually becomes more severe and irritating.

After 1-2 weeks, the second, or paroxysmal stage, begins. The patient has bursts, or paroxysms, of numerous, rapid coughs, apparently due to difficulty expelling thick mucus from the tracheobronchial tree. At the end of the paroxysm, a long inspiratory effort is usually accompanied by a characteristic whoop. In younger infants, periods of apnea may follow the coughing spasms, and the patient may become cyanotic (turn blue).

In the convalescent stage, recovery is gradual. The cough becomes less paroxysmal and disappears over 2 to 3 weeks. However, paroxysms often recur with subsequent respiratory infections for many months after the onset of pertussis. Fever is generally minimal throughout the course of pertussis.

Pneumonia is a relatively common complication (reported 21.7% of cases in developed countries); otitis, hemorrhages (subconjunctival petechiae and epistaxis), convulsions, encephalopathies and death occur more rarely. The disease lasts 4 to 8 weeks. Complications are more frequent and severe in younger infants. Older persons (i.e. adolescent and adults), and those partially protected by the vaccine usually have milder disease.

Clinical case definition

- A case diagnosed as pertussis by a physician, or
- A person with a cough lasting at least 2 weeks with at least one of the following symptoms:
  - Paroxysms (i.e. fits) of coughing
  - Inspiratory “whooping”
  - Post-tussive vomiting (i.e. vomiting immediately after coughing)

Case classification

Clinical case: A case that meets the clinical case definition.
Confirmed case: A clinical case that is laboratory confirmed (isolation of Bordetella pertussis, detection of genomic sequences by PCR, or positive paired serology).

1.8.2 Infectious agent

Bacterium: Bordetella pertussis
1.8.3 Occurrence

Pertussis in Iraq\textsuperscript{16}.

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1.8.4 Reservoir

Humans are the only hosts.

1.8.5 Mode of transmission

- Primarily by direct contact with discharges from respiratory mucous membranes of infected persons via the airborne route.
- Even though the disease may be milder in older persons, these infected persons may transmit the disease to other susceptible persons, including non-immunized or under-immunized infants. Adults are often found to be the first case in a household with multiple pertussis cases.

1.8.6 Incubation period

Usually 7-10 days, rarely more than 14 days.

1.8.7 Period of communicability

Pertussis is highly communicable in the early catarrhal stage. Communicability gradually decreases after the onset of the paroxysmal cough. Untreated patients may be contagious for up to 3 weeks after the onset of paroxysmal cough in the absence of treatment or up to 5 days after onset of treatment.

1.8.8 Susceptibility and resistance

Anyone who has not had pertussis previously or who has not received the pertussis vaccine can get the disease. Immunity following disease or vaccination is not lifelong. Older children, adolescents and adults can become susceptible to pertussis five-to 10-years after their last dose of pertussis-containing vaccine.

1.8.9 Methods of control\textsuperscript{17}

1.8.9a Preventive measures (Immunization)

\textsuperscript{16} http://apps.who.int/immunization_monitoring/en/globalsummary/timeseries/tsincidencebycountry.cfm?C=IRQ
\textsuperscript{17} http://www.cdc.gov/pertussis/clinical/features.html

The administration of vaccines is the most rational approach to pertussis control. Active primary immunization against *B. pertussis* infection with whole-cell vaccine (wP) is recommended in association with the administration of diphtheria and tetanus toxoids (DTP). No single antigen pertussis vaccine is available. Although the use of acellular vaccines (aP) is less commonly associated with adverse reactions, price considerations affect their use, and wP vaccines are the vaccines of choice for most countries, including Iraq. In general, pertussis vaccine is not given to persons 7 years of age or older, since reactions to the vaccine (convulsions, collapse, high temperature) may be increased in older children and adults. The efficacy of the vaccine in children who have received at least 3 doses is estimated to be 80%: protection is greater against severe disease and begins to wane after about 3 years.

1.8.9b Control measures (Case management)

Erythromycin or erythromycin estolate or – in case of allergies to erythromycin – trimethoprim-sulfamethoxazole (contraindicated during pregnancy) should be administered for 7-14 days to all cases and close contacts of persons with pertussis, regardless of age and vaccination status. Doses recommended by the Iraqi Ministry of Health are 40 mg/kg/day for children and 1 g/day for adults. Drug administration both (1) modifies the course of illness (if initiated early), and (2) eradicates the organism from secretions, thereby decreasing communicability. Symptomatic treatment and supportive case-management.

1.8.9c Epidemic measures

The highly contagious nature of the disease leads to large numbers of secondary cases among non-immune contacts. Prophylactic antibiotic treatment (erythromycin) in the early incubation period may prevent disease, but difficulties of early diagnosis, costs involved and concerns related to the occurrence of drug resistance all limit prophylactic treatment to selected individual cases.

Priority must be given to:

- Protecting children less than 1 year old and pregnant females in the last 3 weeks of pregnancy because of the risk of transmission to the newborn.
- Stopping infection among household members, particularly if there are children aged less than 1 year and pregnant women in the last 3 weeks of pregnancy.

The strategy relies on chemoprophylaxis of contacts within a maximum delay of 14 days following the first contact with the index case. Index cases must avoid contact with daycare centers, schools and other places regrouping susceptible individuals for up to 5 days after the beginning of treatment or up to 3 weeks after onset of paroxysmal cough, or till the end of cough, whichever comes first. All contact cases must have their immunization status verified and brought up to date.
### 1.8.10 Management of the disease

- **Case management:** Antibiotic treatment (Azithromycin, Clarithromycin, Erythromycin, TMP-SMX). Note: cases should be commenced on antibiotic therapy on clinical suspicion to reduce the risk of transmission (only commence if within 21 days of onset of symptoms i.e. coryza). Exclude from childcare, pre-school, school or work until 5 days of antibiotic treatment is complete.

- **Contact Management:** Identify high risk household contacts and provide chemoprophylaxis
  - Children <24 months with less than 3 doses of pertussis vaccine received (also provide chemoprophylaxis to all household members).
  - Pregnant women in the last month of pregnancy (also provide chemoprophylaxis to all household members).

Ref: [http://www.cdc.gov/pertussis/clinical/features.html](http://www.cdc.gov/pertussis/clinical/features.html)

Epidemiology and Prevention of Vaccine-Preventable Diseases The Pink Book: Course Textbook - 12th Edition (April 2011)

A young boy coughing due to pertussis

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1.9 Acute Poliomyelitis

ICD-10 A80

1.9.1 Identification

Poliomyelitis is a highly contagious disease caused by poliovirus.

Clinical features:
1. **Unapparent infection without symptoms**: Up to 95% of all polio infections are unapparent or sub clinical. Estimates of ratio of unapparent to paralytic illness vary from 50:1 to 1000:1 (usually 200:1).
2. **Minor illness (abortive poliomyelitis)**: Approximately 5% of polio infections consist of nonspecific illness without clinical or laboratory evidences of central nervous system invasion and is characterized by complete recovery in less than one week. Three syndromes observed with this form of poliovirus infection, which are upper respiratory tract infection (sore throat and fever), gastrointestinal disturbances (nausea, vomiting, abdominal pain, constipation or, rarely diarrhea), and influenza-like illness. These syndromes are indistinguishable from other viral illnesses.
3. **Non-paralytic poliomyelitis**: Non-paralytic aseptic meningitis usually following several days after a prodrome similar to that of minor illness occur in 1%-2% of polio infections. These symptoms will last from 2 to 10 days followed by complete recovery.
4. **Paralytic poliomyelitis**: Less than 2% of all polio infections result in a flaccid paralysis. Paralytic symptoms generally begin 1 to 10 days after prodromal symptoms and progress for 2 to 3 days. Generally, no further paralysis occurs after the temperature returns to normal. The prodrome may be biphasic, especially in children, with initial minor symptoms separated by a 1 to 7 days period from more major symptoms. Additional prodromal signs and symptoms can include a loss of superficial reflex initially increased deep tendon reflexes and severe muscle aches and spasms in the limbs or back. The illness progresses to flaccid paralysis with diminished deep tendon reflexes, which reaches plateau without change for days or weeks and is usually asymmetrical. Patients do not experience sensory loss or changes in cognition. Many persons with paralytic poliomyelitis recover completely and, in most, muscle function returns to some degree. Patients with weakness or paralysis 12 months after onset will usually be left with permanent residua. Depending on the sites of paralysis, poliomyelitis can be classified as spinal, bulbar, or spino-bulbar disease.

1.9.2 Infectious agent

Poliovirus is a member of the enterovirus subgroup, family Picornaviridae. There are three poliovirus serotypes (P1, P2, and P3). There is minimal heterotypic immunity between the three serotypes.
1.9.3 Occurrence

Polio in Iraq

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Above refers to all polio cases (indigenous or imported), including polio cases caused by vaccine derived polio viruses (VDPV); it does not include cases of vaccine-associated paralytic polio (VAPP) and cases of non polio acute flaccid paralysis (AFP).

1.9.4 Reservoir

Humans, usually persons with an inapparent infection. Enteroviruses are transient inhabitants of the gastrointestinal tract, and are stable at acid PH.

1.9.5 Mode of transmission

Fecal-oral transmission: transmitted when people drink water or eat food contaminated by feces (or stools) which carry the virus. Can also be transmitted through droplet spread of respiratory secretions of an infected person. Infants shedding virus in the feces after having received OPV have been the source of exposure for susceptible adults giving child care.

1.9.6 Incubation period

Typically 6 to 20 days, range 3 to 35 days.

1.9.7 Period of communicability

Persons with polio are most contagious shortly before and after the onset of symptoms. The virus is present in respiratory secretions for about a week and in the feces for up to six weeks after onset of illness. Persons with asymptomatic infections are also communicable.

1.9.8 Susceptibility and resistance

Paralytic poliomyelitis is fatal in 2%-10% of cases. More than 95% of vaccinated individuals develop long-lasting immunity. Heat, formaldehyde, chlorine, and ultraviolet light rapidly inactivate the poliovirus. Overcrowding and poor sanitation provide opportunities for exposure to infection.

1.9.9 Methods of prevention

**Oral Poliovirus Vaccine (OPV):** Trivalent OPV contains live attenuated strains of all three serotypes of poliovirus. Live attenuated polioviruses replicate in the intestinal mucosa and lymphoid cells, and in lymph nodes that drain the intestine. Vaccine viruses may spread from the

21 http://www.polioeradication.org/Polioandprevention.aspx
recipient to contacts. Persons coming in contact with fecal material of a vaccinated person may be exposed and infected with vaccine virus. After complete primary vaccination with three doses of OPV, > 95% of recipients develop long-lasting immunity to all three poliovirus types. Approximately 50% of vaccine recipients develop antibody to all three serotypes after a single dose of OPV. OPV induces immunity of the gastrointestinal tract that provides a substantial degree of resistance to re-infection with poliovirus. It contains neomycin and streptomycin.

1.9.10 Management of the disease

- One in 200 infections leads to irreversible paralysis, usually in the legs. This is caused by the virus entering the blood stream and invading the central nervous system. As it multiplies, the virus destroys the nerve cells that activate muscles. The affected muscles are no longer functional and the limb becomes floppy and lifeless – a condition known as acute flaccid paralysis (AFP). All cases of acute flaccid paralysis (AFP) among children under fifteen years of age are reported and tested for poliovirus within 48 hours of onset.

- Around 40% of people who survive paralytic polio may develop additional symptoms 15–40 years after the original illness. These symptoms – called post-polio syndrome – include new progressive muscle weakness, severe fatigue and pain in the muscles and joints.

- There is no cure for polio, only treatment to alleviate the symptoms. Heat and physical therapy is used to stimulate the muscles and antispasmodic drugs are given to relax the muscles. While this can improve mobility, it cannot reverse permanent polio paralysis.

- Polio can be prevented through immunization. Polio vaccine, given multiple times, almost always protects a child for life.

Ref: [http://www.polioeradication.org/Polioandprevention.aspx](http://www.polioeradication.org/Polioandprevention.aspx)

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A child with a deformity of her right leg due to polio
1.10 Rabies

ICD-10 A82

1.10.1 Identification

A fatal acute viral disease caused by rabies virus, a *rhabdovirus* of the genus *Lyssavirus*, whose first symptoms may be apprehension, headache and indefinite sensory changes, fever, malaise, water and air phobia, and paralysis. It is transmitted in the saliva of infected animals, most commonly through bites. Rabies is an acute encephalomyelitis that almost progresses to coma or death within 10 days after the first symptoms.

**Case classification**

- *Suspected:* A case that is compatible with the clinical description.
- *Probable:* Not applicable.
- *Confirmed:* A suspected case by especial laboratory investigation

**Laboratory criteria for diagnosis**

One of the following:

- Detection of rabies viral antigens by direct florescent antibody (FA) in clinical specimens, preferably brain tissue (collected post mortem).
- Detection by FA on skin or corneal smear (collected ante mortem).
- FA positive after inoculation of brain tissue, saliva or CSF in cell culture, in mice or in sucking mice.
- Detectable rabies-neutralizing antibody titer $\geq 10.5$ in the CSF of an unvaccinated person.
- Identification of viral antigens by PCR on fixed tissue collected post mortem or in a clinical specimen (brain tissue or skin, cornea or saliva).
- Isolation of rabies virus from clinical specimen and confirmation of rabies viral antigens by direct fluorescent antibody testing.

1.10.2 Infectious agent

A *rhabdovirus* of the genus *Lyssavirus*.

1.10.3 Occurrence

**Cases of Rabies, 2000-2009**

<table>
<thead>
<tr>
<th>Year</th>
<th>2000</th>
<th>2001</th>
<th>2002</th>
<th>2003</th>
<th>2004</th>
<th>2005</th>
<th>2006</th>
<th>2007</th>
<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases</td>
<td>14</td>
<td>7</td>
<td>11</td>
<td>12</td>
<td>21</td>
<td>29</td>
<td>18</td>
<td>24</td>
<td>16</td>
<td>27</td>
<td>29</td>
<td>8</td>
</tr>
</tbody>
</table>
1.10.4 Reservoir

Rabies is primarily a disease of animals, particularly wild and domestic canine species, cats, bats, and other biting animals.

1.10.5 Mode of transmission

- It is transmitted by the virus-laden saliva of a rabid animals introduced via bite or scratch.
- Dogs and cats are the main urban vectors.
- Person to person transmission via saliva is theoretically possible but has never been documented.

1.10.6 Incubation period

Usually two to eight weeks, but can occur from five days to over a year depending on factors such as severity, site of wound and infective dose. Very long incubation periods of up to six years or more have been reported.

1.10.7 Period of communicability

Rabies virus is present in saliva, CSF, and neurologic tissues of infected patients who are in the final (clinical) stage of disease. Rabid dogs, cats are considered communicable no more than 10 days prior to symptom onset. Little or nothing is known about how early communicability starts in other species, including humans.

1.10.8 Susceptibility and resistance

All age groups are susceptible, however, most common in people younger than 15 years.

1.10.9 Methods of control\textsuperscript{24}

- Report to local health authority, CDC.
- Post-exposure vaccination of bitten people at day (0, 3, 7, 14, 28) of bite.
- Vaccination of the domestic dogs and cats by veterinary hospitals.
- Contact isolation for respiratory secretions for duration of illness.
- Stray dog control.
- Take contact precautions (gloves, gown and mask) when nursing a case of rabies.

1.10.10 Management of the disease

- **Treatment after exposure:** Effective treatment soon (within a few days, but as soon as possible) after exposure to rabies can prevent the onset of symptoms and death. Post-exposure prevention consists of local treatment of the wound, administration of rabies immunoglobulin (if indicated), and immediate vaccination.

- **Local treatment of the wound:** Removing the rabies virus at the site of the infection by chemical or physical means is an effective means of protection. Therefore, prompt local treatment of all bite wounds and scratches that may be contaminated with rabies virus is important. Recommended first-aid procedures include immediate and thorough flushing and washing of the wound for a minimum of 15 minutes with soap and water, detergent, povidone iodine or other substances that kill the rabies virus.

- **Recommended treatment:** The recommended post-exposure prophylaxis depends on the type of contact with the suspected rabid animal (see table).

Table: Recommended post-exposure prophylaxis for rabies infection

<table>
<thead>
<tr>
<th>Category of exposure to suspect rabid animal</th>
<th>Post-exposure measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category I – touching or feeding animals, licks on intact skin (i.e. no exposure)</td>
<td>None</td>
</tr>
<tr>
<td>Category II – nibbling of uncovered skin, minor scratches or abrasions without bleeding</td>
<td>Immediate vaccination and local treatment of the wound</td>
</tr>
<tr>
<td>Category III – single or multiple transdermal bites or scratches, licks on broken skin; contamination of mucous membrane with saliva from licks, exposures to bats.</td>
<td>Immediate vaccination and administration of rabies immunoglobulin; local treatment of the wound</td>
</tr>
</tbody>
</table>

Other factors that should be taken into consideration when deciding whether to initiate post-exposure prevention include:

- the likelihood of the implicated animal being rabid
- the clinical features of the animal and its availability for observation and laboratory testing.

In developing countries, the vaccination status of the suspected animal alone should not be considered when deciding whether to initiate prophylaxis or not.

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1.11 Rubella (German measles)

ICD-10 B06: Rubella (German Measles)
ICD-10 P35: Congenital Rubella Syndrome (CRS)

1.11.1 Identification

Rubella is a mild febrile viral disease with a diffuse punctuate and maculopapular rash. Up to 50% of persons with acquired rubella have asymptomatic infections. Young children usually have little or no prodrome, while adolescents and adults often report 1–5 days of low grade fever, malaise, and anorexia. Lymphadenopathy (usually suboccipital, postauricular, and posterior cervical) is a major clinical manifestation and may last several weeks. Fever rarely persists beyond the first day of rash.

The maculopapular rash appears first on the face and spreads down the body. Lesions are pink and rarely coalesce. The rash of acquired rubella typically lasts 3 days, spreading and fading more quickly than the rash caused by measles.

It can cause congenital rubella syndrome (CRS) in the infant born to a pregnant woman infected with rubella. CRS includes low birth weight, eye defects (cataracts, microphthalmia, glaucoma, retinopathy), sensorineural deafness, cardiac defects (patent ductus arteriosus, peripheral pulmonary artery stenosis), central nervous system defects (microencephaly, mental retardation), hepatitis, thrombocytopenic purpura, splenomegaly, and bone lesions. Deafness is the most common manifestation of CRS, and is sometimes the only manifestation.

Case classification

Suspect: any generalized maculopapular rash with fever, lymphadenopathy
Probable: a case that meets the clinical case definition, has no or noncontributory serological or virological testing, and is not epidemiologically linked to a laboratory confirmed case.
Confirmed: a case that is laboratory confirmed (isolation of rubella virus or significant rise in rubella antibody level by standard serologic assay, or positive serologic test for rubella IgM antibody) or that meets the clinical case definition and is epidemiologically linked to a laboratory - confirmed case.

1.11.2 Infectious agent

Rubella virus (family togaviridae; genus Rubivirus).
1.11.3 Occurrence

In the absence of generalized immunization rubella occurred worldwide at endemic level with epidemics every 5-9 years. Large rubella epidemics resulted in very high levels of morbidity.

Rubella in Iraq\(^{26}\).

<table>
<thead>
<tr>
<th>Year</th>
<th>2000</th>
<th>2001</th>
<th>2002</th>
<th>2003</th>
<th>2004</th>
<th>2005</th>
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<th>2008</th>
<th>2009</th>
<th>2010</th>
<th>2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases</td>
<td>1612</td>
<td>91</td>
<td>-</td>
<td>-</td>
<td>383</td>
<td>99</td>
<td>72</td>
<td>51</td>
<td>110</td>
<td>167</td>
<td>15</td>
<td>20</td>
</tr>
</tbody>
</table>

1.11.4 Reservoir

Infected humans.

1.11.5 Mode of transmission

- Contact with nasopharyngeal secretions of infected person.
- Infection by droplet spread or direct contact with patients.
- Infants with CRS shed large quantities of virus in their pharyngeal secretions and urine and serve as a source of infection to their contacts.

1.11.6 Incubation period

14-21 days.

1.11.7 Period of communicability

1 week before and at least 4 days after onset of rash; infants with CRS may shed virus for months after birth.

1.11.8 Susceptibility and resistance

- Immunity is usually permanent after natural infection and thought to be long-term, probably lifelong.
- Immunity after immunization, but this may depend on contact with endemic cases.
- Infants born to immune mothers are ordinarily protected for 6-9 months, depending on the amount of maternal antibodies acquired transplacentally.

1.11.9 Methods of control

1.11.9a Preventive measures

- Educate the general public on modes of transmission and stress the need for rubella immunization.
- WHO recommend that all countries assess their rubella situation and, if appropriate, make plans for the introduction of rubella vaccine.
- In case of infection with wild rubella virus early in pregnancy, culturally appropriate counseling should be provided. Abortion may be considered in those countries where this is an option.
- Immune globulin (IG) given after exposure early in pregnancy may not prevent infection or viremia, but it may modify or suppress symptoms.

1.11.9b Control measures

- Report to local health authority: All cases of rubella and of CRS should be reported.
- Isolation: In hospitals, patients suspected of having rubella should be managed under contact isolation precautions; attempts should be made to prevent exposure of non-immune pregnant women.
- Concurrent disinfection: Not applicable.
- Quarantine: Not applicable.
- Immunization of contacts: will not necessarily prevent infection or illness.
- Investigation of contacts and source of infection: identify pregnant female contacts, especially those in the first trimester. Such contacts should be tested serologically for susceptibility or early infection (IgM antibody) and advised accordingly.

1.11.9c Epidemic measures

- Prompt reporting of all confirmed and suspected cases.
- The medical community and general public should be informed about rubella epidemics in order to identify and protect susceptible pregnant women.

1.11.10 Management of the disease

- No specific treatment is available. Symptomatic/supportive treatment should be given.

Ref: Epidemiology and Prevention of Vaccine-Preventable Diseases The Pink Book: Course Textbook - 12th Edition (April 2011)
Rash of rubella on skin of child's back. Distribution is similar to that of measles but the lesions are less intensely red.

http://phil.cdc.gov/PHIL_Images/03052002/00002/PHIL_712_lores.jpg
1.12 Meningococcal Disease

ICD-10 A39

1.12.1 Identification

Clinical case definition
Invasive meningococcal disease most commonly presents as meningitis, meningococcemia, or both. Symptoms of meningococcal meningitis include sudden onset of fever (>38.5 °C rectal or >38.0 °C axillary) and one or more of the following: nausea, vomiting, photophobia, neck stiffness, altered mental status, other meningeal sign. Symptoms of meningococcemia (septicemia) include acute onset of fever often accompanied by hypotension and shock, and may include a petechial or purpuric rash, purpura fulminans, and multiorgan failure.

In patients under one year of age, suspect meningitis when fever is accompanied by bulging fontanelles.

Case classification
- **Suspected**: a case that meets the clinical case definition
- **Probable**: a suspected case as defined above, and turbid CSF (with or without positive Gram-stain), or ongoing epidemic and epidemiological link to a confirmed case.
- **Confirmed**: a suspected or probable case with laboratory confirmation (positive CSF antigen detection or positive culture)

1.12.2 Infectious agent

Bacterium: *Neisseria meningitidis* serogroups A, B, C, Y, W135

1.12.3 Occurrence

No data available.

1.12.4 Reservoir

Humans are the only reservoirs.

1.12.5 Mode of transmission

Transmission occurs through respiratory droplets or by direct contact with nasopharyngeal secretions from a colonized person – symptomatic or otherwise.

1.12.6 Incubation period

Between 2-10 days, usually 4 days.
1.12.7 Period of communicability

The most important source of infection are asymptomatic carriers. Persons can transmit the organism to others as long as meningococci are present in nasal or pharyngeal secretions. Cases should be considered infectious from the time they are exposed until 24 hours after initiation of treatment or chemoprophylaxis with appropriate antibiotics. Contacts exposed to the patient 7-10 days or more before his/her onset of illness are not at significantly increased risk.

1.12.8 Susceptibility and resistance

Close contacts of a case are at increased risk of becoming colonized/infected and developing illness. The attack rate for household contacts of cases is 500–800 times the rate that for the general population. Risk of disease in close contacts is highest during the 10-day period following exposure.

1.12.9 Methods of control

1.12.9a Preventive measures

- **Vaccination**: to prevent secondary cases around a sporadic case of meningococcal disease, vaccine can be used for close contacts of patients with meningococcal disease due to A, C, Y, or W135 serogroups.

- **Chemoprophylaxis**: the aim of chemoprophylaxis is to prevent secondary cases by eliminating nasopharyngeal carriage. To be effective in preventing secondary cases, chemoprophylaxis must be initiated as soon as possible (i.e. not later than 48 hours after diagnosis of the case). Its use should be restricted to close contacts of a case, which are defined as:
  - Household members (i.e. persons sleeping in the same dwelling as the case)
  - Institutional contacts who shared sleeping quarters (i.e. for boarding-school pupils, roommates; for military camps, persons sharing a barracks);
  - Nursery school or childcare centre contacts (i.e. children and teachers who share a classroom with the case);
  - Others who have had contact with the patient’s oral secretions through kissing or sharing of food and beverages.

The drugs recommended by the Iraqi Ministry of Health are Rifampin (children and adults) or Ciprofloxacin (adults only).

1.12.9b Control measures

Meningococcal disease (either meningitis or septicemia) is potentially fatal and should always be viewed as a medical emergency. The Iraqi Ministry of Health recommends that any suspected case should be referred to a hospital immediately. Admission to a hospital or health centre is necessary for diagnosis by lumbar puncture and CSF.

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examination. Lumbar puncture must be done as soon as meningitis is suspected, prior to starting antibacterials. As infectivity of patients is moderate and disappears quickly following antimicrobial treatment, isolation of the patient is not necessary.

Antimicrobial therapy must be instituted as soon as possible after lumbar puncture (without waiting for laboratory results), and should be combined with supportive treatment. Initial antimicrobial therapy should be effective against the three major causes of bacterial meningitis until bacteriological results are available. Once diagnosis of meningococcal disease has been established, many antimicrobials can be used: either penicillin or ampicillin is the drug of choice. Chloramphenicol is a good and inexpensive alternative. The third-generation cephalosporins, Ceftriaxone and Cefotaxime, are excellent alternatives but are considerably more expensive. A seven-day course is still the general rule for the treatment of meningococcal disease (beyond the neonatal period). The long-acting (oily) form of chloramphenicol has also been shown to be effective.

1.12.9c Epidemic measures

- **Vaccination**: a mass vaccination campaign, if appropriately carried out, is able to halt an epidemic of meningococcal disease. Laboratory diagnosis and confirmation of epidemic serogroups will guide the type of vaccine needed, either meningococcal polysaccharide bivalent A/C (if serogroup A or C is confirmed as the epidemic serogroup), or meningococcal polysaccharide tetravalent vaccine A/C/Y/W135 (if serogroup W135 or Y is confirmed). Vaccination will be concentrated in the area where the epidemic is maximal.

- **Refugee camp population**: Following confirmation (serogroup identified) of two cases, mass vaccination is recommended if the serogroup/s identified is/are included in either the bivalent (A/C) or tetravalent (A/C/Y/W135) vaccine. At risk populations (e.g. 2-30 years of age) should be given priority.

- **General population**: If an outbreak is suspected, vaccination should only be considered after careful investigation (including confirmation and serogroup identification) and the assessment of the population group at highest risk.

- **Chemoprophylaxis**: chemoprophylaxis of contacts of meningitis patients is NOT warranted during an epidemic for several reasons. In small clusters or outbreaks among closed populations (e.g. extended household, boarding schools), chemoprophylaxis may still be appropriate.

- **Diagnosis**: as the flood of patients could make the routine use of lumbar puncture to confirm meningitis impossible, every suspected case of meningitis should be considered and treated as one of meningococcal meningitis.

- **Treatment**: simplified treatment protocols are appropriate: long-acting oily chloramphenicol intramuscularly (100 mg/kg up to 3 grams in a single dose) is the drug of choice for all age groups, particularly in areas with limited health facilities.
1.12.10 Management of the disease

- Meningococcal disease is potentially fatal and should always be viewed as a medical emergency. Admission to a hospital or health centre is necessary, although isolation of the patient is not necessary. Appropriate antibiotic treatment must be started as soon as possible, ideally after the lumbar puncture has been carried out if such a puncture can be performed immediately. If treatment is started prior to the lumbar puncture it may be difficult to grow the bacteria from the spinal fluid and confirm the diagnosis.
- A range of antibiotics can treat the infection, including penicillin, ampicillin, chloramphenicol and ceftriaxone. Under epidemic conditions in Africa in areas with limited health infrastructure and resources, oily chloramphenicol or ceftriaxone are the drugs of choice because a single dose has been shown to be effective on meningococcal meningitis.

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1.13 Malaria

ICD-10 B50: Falciparum
ICD-10 B51: Vivax
ICD-10 B52: Malaria
ICD-10 B53: Ovale
ICD-10 B54: Unspecified Malaria

1.13.1 Identification

A parasitic infections disease; with 4 human types of malaria causing symptoms sufficiently similar to make species differentiation impossible without laboratory studies. The most prominent feature of malaria is fever. Classic descriptions of fever with a regular recurring pattern every two or three days is not usually present when the disease begins. Irregular fever also may occur due to mixed infections and ineffective use of prophylactic drugs and partial treatment. Patients commonly feel well on the days when fever is absent. A presumptive diagnosis of malaria should be made for any person with a high fever who has been to a malarious area until proved otherwise, particularly with recent travel. Early diagnosis with prompt appropriate treatment is essential as malaria can be a fatal disease. If the initial blood film is negative for malarial parasites it should be repeated within 12–24 hours and preferably when the temperature is rising. One negative test does not exclude the diagnosis, particularly if the patient has taken antibiotics which may result in partial treatment of the infection.

Clinical manifestations

The rapidly rising temperature is commonly associated with shaking chills, muscle pains, back pain, nausea and headache, and the episode frequently ends with profuse sweating. Other symptoms may include confusion or other neurological signs, diarrhea, dark urine, jaundice, cough and respiratory distress.

Complications

The following severe complications may occur, usually with *P. falciparum* infections: coma, acute encephalopathy, cerebral edema, vomiting, renal failure, severe anemia, thrombocytopenia, pulmonary edema, shock, acidosis, coagulation defects, respiratory failure, liver failure and death. Atypical presentations can occur which predominantly involve a diarrheal illness and have resulted in delayed diagnosis and death. Other infections such as the bacterial infection typhoid fever may occur concurrently. These should be looked for, especially if the patient fails to respond well to appropriate treatment. Individuals, who are partially immune or have been taking anti-malarial chemoprophylaxis, may show an atypical clinical picture with wide variations in the incubation period. Malaria due to species other than *P. falciparum* is generally not life threatening except in the very young, very old and those with immunodeficiency or other concurrent disease.

Case definition

A patient exposed to malaria who is hospitalized for a febrile disease with no obvious cause.
Case classification

**Suspected case**: Detection of Plasmodium species by rapid diagnostic antigen testing without confirmation by microscopy or nucleic acid testing in any person (symptomatic or asymptomatic)

**Confirmed case**: the confirmation is through:

- Identification of malaria parasites in blood films. Repeated microscopic examinations every 12–24 hours may be necessary because the blood density of parasites varies and parasites are often not demonstrable in films from patients recently or actively under treatment.
- Detection of plasmodia antigens in the blood by PCR is the most sensitive method, but is not generally available in diagnostic laboratories.
- Antibodies, demonstrable by IFA, may appear after the first week of infection but may persist for years, indicating past malarial experience; thus antibody determinations are not helpful for diagnosis of current illness.

1.13.2 Infectious agent

1. *P. Falciparum*: the most serious malarial infection; eradicated in Iraq since the 1950s.
2. *P. Vivax*: most common in Iraq
3. *P. Ovale*
4. *P. Malaria*

1.13.3 Occurrence

Iraq now can be consider as free of malaria since 2008 in the phase of elimination, and we are waiting the certification for that from WHO, there is strong supervision efforts by members of malarial section of CDC –Baghdad to the all parts of Iraq especially on the northern and southern parts.

1.13.4 Reservoir

Humans are the main reservoir for the parasite.

1.13.5 Modes of transmission

Transmission occurs by the bite of infected anopheles mosquitoes (female).

1.13.6 Incubation period

<table>
<thead>
<tr>
<th>Malarial species</th>
<th>Incubation period</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>P. falciparum</em></td>
<td>8-14 days</td>
</tr>
<tr>
<td><em>P. vivax and P. ovale</em></td>
<td>12-18 days</td>
</tr>
<tr>
<td><em>P. malariae</em></td>
<td>8-14 days</td>
</tr>
</tbody>
</table>
1.13.7 Period of communicability

1) Plasmodium parasites must undergo developmental changes in a competent mosquito host before being passed back to another human; this takes from a week and a month. Human are communicable to mosquitoes when gametocytes are present in blood.
2) Untreated or insufficiently treated patients may be a source of mosquito infection for several years in *P. malariae*, up to 5 years in *P. vivax*, and generally not more than 1 year in *P. falciparum* malaria; the mosquito remains infective for life.
3) Transfusional transmission may occur as long as asexual forms remain in the circulating blood (with *P. malariae*, up to 40 years or longer).
4) Stored blood can remain infective for at least a month.

1.13.8 Susceptibility and resistance

Susceptibility is universal except in humans with specific genetic traits. In Iraq, genetic traits that may modify disease expression include 1) sickle cell, 2) thalassemias, 3) glucose-6-phosphate dehydrogenase (G6PD) deficiency. The immune-suppressed persons living in endemic areas appear to be at increased risk of more frequent and higher density infections, and may show decreased response to any malarial therapy.

1.13.9 Methods of control

1.13.9a Preventive measures

1. Avoid being bitten by mosquitoes, especially between dusk and dawn (personal protection) is the first line of defense against malaria. Other measures include: wearing long cloth, insect repellents, sprays, removing mosquito breeding places or water collections near the house and use insecticide-treated mosquito nets (ITNs).
2. Chemo-prophylactic: given for the travelers going to known malarial area or countries included (Iran, Pakistan, Philippines, India, Thailand and other south east of Asia, all African countries except the north part of Africa and Yemen).
3. Immediately seeking diagnosis and treatment if any suggestive sign and symptom of malaria develops.

1.13.9b Control measures

Isolation of the case is not required. Generally if the species cannot be identified with confidence, the patient should be treated as for the most serious infection with *P. falciparum*. This can be applied especially on the imported cases of malaria. On the other hand, as we know, the existing species of malaria in Iraq is *P. vivax* so the regime of treatment is as shown below: Radical treatment of all positive cases by Chloroquine tablet; base dosage is:
<table>
<thead>
<tr>
<th>1&lt;sup&gt;st&lt;/sup&gt; day</th>
<th>2&lt;sup&gt;nd&lt;/sup&gt; day</th>
<th>3&lt;sup&gt;rd&lt;/sup&gt; day</th>
<th>4&lt;sup&gt;th&lt;/sup&gt; day</th>
<th>17&lt;sup&gt;th&lt;/sup&gt; day</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 tabs. Initially then 2 tabs after 6 hrs.</td>
<td>2 tabs</td>
<td>2 tabs</td>
<td>Primaquine tab. (15) mg one tab/day for 14 day</td>
<td>Last dose of Primaquine tab in order to reduces the risk of relapses of disease</td>
</tr>
</tbody>
</table>

1.13.9c Epidemic measures

We need rapid and vigorous action and effective treatment of all cases; as mass treatment, full coverage vector control measures should be instituted. Usually, evening fogging, indoor residual spraying is preferred because of its rapid effect; this may be followed by the use of insecticide treated bed nets and anti-larval measures.

1.13.9d Disaster implications

Disasters may lead to malaria epidemics as a result of population movements, ecological changes, breakdown of health services, and other factors. So we need urgent or emergency interventions to be made in the malarial sections of the provinces and apply control measures including early effective treatment and vector control, insecticide-treated nets, indoor residual spraying, fogging - evening thermal and cold type, distribution of mosquito nets and health education.

1.13.9e International measures

1) **Important international measures include the following:**
   a) Disinsectization of aircraft before boarding passengers or in transit, using a residual spray application of an effective insecticide;
   b) Disinsectization of aircraft, ships and other vehicles on arrival if the health authority at the place of arrival has reason to suspect importation of malaria vectors;
   c) Enforcing and maintaining rigid anti-mosquito sanitation within the mosquito flight range of all ports and airports.

2) **In special circumstances:**
   Administer antimalarial drugs to potentially infected migrants, refugees, seasonal workers and persons taking part in periodic mass movement into an area or country where malaria has been eliminated.

1.13.10 Management of the disease

- The best available treatment, particularly for *P. falciparum* malaria, is artemisinin-based combination therapy (ACT).
- WHO recommends that all cases of suspected malaria be confirmed using parasite-based diagnostic testing (either microscopy or rapid diagnostic test) before administering

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treatment. Results of parasitological confirmation can be available in 15 minutes or less. Treatment solely on the basis of symptoms should only be considered when a parasitological diagnosis is not possible.

Ring-forms and gametocytes of *Plasmodium falciparum* in human blood

1.14 Measles

ICD-10 B05

1.14.1 Identification

Acute systemic viral infection. The prodromal phase lasts 2–4 days (range 1–7 days). It is characterized by fever, which increases in stepwise fashion, often peaking as high as 103°–105°F. This is followed by the onset of cough, coryza (runny nose), or conjunctivitis. Koplik spots, a rash (enanthem) present on mucous membranes, is considered to be pathognomonic for measles. It occurs 1–2 days before the rash to 1–2 days after the rash, and appears as punctate blue-white spots on the bright red background of the buccal mucosa.

The measles rash is a maculopapular eruption that usually lasts 5–6 days. It begins at the hairline, then involves the face and upper neck. During the next 3 days, the rash gradually proceeds downward and outward, reaching the hands and feet. The maculopapular lesions are generally discrete, but may become confluent, particularly on the upper body. Initially, lesions blanch with fingertip pressure. By 3–4 days, most do not blanch with pressure. Fine desquamation occurs over more severely involved areas. The rash fades in the same order that it appears, from head to extremities.

Complications include diarrhea, dehydration, stomatitis, inability to feed, and bacterial infections (skin and elsewhere).

Clinical case definition
Any person with fever and generalized maculopapular (non-vesicular) rash and cough, coryza (i.e. runny nose) or conjunctivitis (i.e. red eyes). OR
Any person in whom a clinical health worker suspects measles infection.

Case classification
Clinically confirmed: a case that meets the clinical case definition
Laboratory confirmed: (only for outbreak confirmation and during the elimination phase): A case that meets the clinical case definition and is laboratory confirmed (presence of measles-specific IgM antibodies); A case meeting clinical definition and epidemiologically linked by direct contact to a laboratory-confirmed case in which rash onset occurred 7-18 days earlier.

1.14.2 Infectious agent

Measles virus (genus Morbillivirus, family Paramyxoviridae)
1.14.3 Occurrence

Measles in Iraq.\(^{33}\)

<table>
<thead>
<tr>
<th>Year</th>
<th>2000</th>
<th>2001</th>
<th>2002</th>
<th>2003</th>
<th>2004</th>
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In temperate areas, measles disease occurs primarily in late winter and spring.

1.14.4 Reservoir

Humans. Asymptomatic carrier state has not been documented.

1.14.5 Mode of transmission

- Airborne by droplet spread; or
- Direct contact with the nasal and throat secretions of infected persons or via object (e.g. toys) that has been in close contact with an infected person

1.14.6 Incubation period

After infection there is an asymptomatic incubation period of 10-12 days, with a range from 7 to 18 days from exposure to the onset of fever.

1.14.7 Period of communicability

Measles is most infectious from 4 days before the rash until 1-2 days after rash onset.

1.14.8 Susceptibility and resistance

Measles is highly contagious one person can infect 90% of the people close to that person who are not immune. Measles is more severe in malnourished children, particularly those with vitamin A deficiency. The case-fatality rate may be as high as 25%.

1.14.9 Methods of control\(^{34,35}\)

1.14.9a Preventive measures

Iraq has a routine immunization policy which requires a dose of single antigen measles vaccine at 9 months, a dose of MMR (measles-mumps-rubella vaccine) at 15 months, and another dose of MMR at school entry. However, supplementary measles immunization campaigns may be required in order to reduce the risk of a measles outbreak.

\(^{34}\) WHO-recommended standard of selected vaccine preventable diseases; http://www.who.int/immunization_monitoring/diseases/measles_surveillance/en/index.html
\(^{35}\) Epidemiology and Prevention of Vaccine-Preventable Diseases The Pink Book: Course Textbook - 12th Edition (April 2011)
1.14.9b Control measures
For uncomplicated cases: Give Vitamin A immediately upon diagnosis and ensure the child receives a second dose the next day (can be given to mother to administer at home). Advise the parent to treat the child at home (control fever and provide nutritional feeding).

For cases with non-severe eye, mouth or ear complications: Children can be treated at home. Give Vitamin A immediately upon diagnosis and ensure that the child receives a second dose the next day (can be given to mother to administer at home). If pus draining from the ears, clean eyes and treat with 1% tetracycline eye ointment. If mouth ulcers, treat with gentian violet. If pus draining from the ear, clean ear discharge and treat with antibiotics for 5 days (amoxycillin –1st line- or cotrimoxazole-2nd line-, as per national ARI policy and IMCI guidelines). Treat malnutrition and diarrhea, if present, with sufficient fluids and high quality diet.

For cases with severe, complicated measles (any general danger signs, clouding of cornea, deep or extensive mouth ulcers, pneumonia): Refer urgently to hospital. Treat pneumonia with an appropriate antibiotic. If clouding of the cornea or pus draining from the eye, clean eyes and apply 1% tetracycline eye ointment. If the child has any eye signs indicating Vitamin A deficiency (i.e. night blindness, Bitôt spots, conjunctival and corneal dryness, corneal clouding or corneal ulceration), then he or she should receive a third dose of Vitamin A 2-4 weeks later.

1.14.9c Epidemic measures
- Inform the Health Authorities if one or more suspected cases are identified.
- Confirm the suspected outbreak, following WHO guidelines.
- Investigate suspected case: check if it fulfills the case definition, record date of onset, age and vaccination status.
- Confirm the diagnosis: collect blood specimen from 3-5 initial reported cases.
- Assess the extent of the outbreak and the population at risk.
- Implement outbreak response measures:
  - Give priority to proper case management and immunization of groups at highest risk (e.g. children 6 months – 5 years) as soon as possible in neighboring areas not yet affected by the outbreak and where the outbreak is likely to spread.
  - Promote social mobilization of parents in order to assure previously unvaccinated children 6 months – 5 years of age are immunized.
  - The presence of several cases of measles in an emergency setting does not preclude a measles immunization campaign. Even among individuals who have already been exposed to, and are incubating the natural virus, measles vaccine, if given within three days of exposure, may provide protection or modify the clinical severity of the illness.
  - Isolation is not indicated and children should not be withdrawn from feeding programs.
1.14.10 Management of the disease

- Severe complications from measles can be avoided through supportive care that ensures good nutrition, adequate fluid intake and treatment of dehydration with WHO-recommended oral rehydration solution. This solution replaces fluids and other essential elements that are lost through diarrhea or vomiting. Antibiotics should be prescribed to treat eye and ear infections, and pneumonia.
- All children in developing countries diagnosed with measles should receive two doses of vitamin A supplements, given 24 hours apart. This can help prevent eye damage and blindness. Vitamin A supplements have been shown to reduce the number of deaths from measles by 50%.

37 Centers for Disease Control and Prevention's Public Health Image Library
1.15 Tetanus

ICD-10 A35: Tetanus
ICD-10 A33: Neonatal Tetanus

1.15.1 Identification

An acute disease induced by an exotoxin of the tetanus bacillus, which grows anaerobically at the site of an injury. The disease is characterized by painful muscular contractions, primarily of the masseter and neck muscles, secondarily of trunk muscles. A common first sign suggestive of tetanus in older children and adults is abdominal rigidity, though rigidity is sometimes confined to the region of injury. Generalized spasms occur, frequently induced by sensory stimuli; typical features of the tetanic spasm are the position of opisthotonos and the facial expression known as “risus sardonicus.” History of an injury or apparent portal of entry may be lacking. The case-fatality rate ranges from 10% to over 80%, it is highest in infants and the elderly, and varies inversely with the length of the incubation period and the availability of experienced intensive care unit personnel and resources. Attempts at laboratory confirmation are of little help. The organism is rarely recovered from the site of infection, and usually there is no detectable antibody response.

1.15.2 Infectious agent

*Clostridium tetani*, the tetanus bacillus.

1.15.3 Occurrence

The disease is more common in agricultural regions and in areas where contact with animal excreta is more likely and immunization is inadequate. Parenteral use of drugs by addicts, particularly intramuscular or subcutaneous use, can result in individual cases and occasional circumscribed outbreaks. In rural and tropical areas people are especially at risk, and tetanus neonatorum is common. The disease is sporadic and relatively uncommon in most industrial countries.

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Tetanus (total) in Iraq\(^{38}\)

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Neonatal Tetanus in Iraq\(^{39}\).


1.15.4 Reservoir

Intestines of horses and other animals, including humans, in which the organism is a harmless normal inhabitant. Soil or fomites contaminated with animal and human feces. Tetanus spores, ubiquitous in the environment, can contaminate wounds of all types.

1.15.5 Mode of transmission

Tetanus spores are usually introduced into the body through a puncture wound contaminated with soil, street dust or animal or human feces; through lacerations, burns and trivial or unnoticed wounds; or by injected contaminated drugs (e.g. street drugs). Tetanus occasionally follows surgical procedures, which include circumcision and abortions performed under unhygienic conditions. The presence of necrotic tissue and/or foreign bodies favors growth of the anaerobic pathogen. Cases have followed injuries considered too trivial for medical consultation.

1.15.6 Incubation period

Usually 3–21 days, although it may range from 1 day to several months, depending on the character, extent and location of the wound; average 10 days. Most cases occur within 14 days. In general, shorter incubation periods are associated with more heavily contaminated wounds, more severe disease and a worse prognosis.

1.15.7 Period of communicability

No direct person-to-person transmission.

1.15.8 Susceptibility and resistance

Susceptibility is general. Active immunity is induced by tetanus toxoid and persists for at least 10 years after full immunization; transient passive immunity follows injection of tetanus immune globulin (TIG) or tetanus antitoxin (equine origin). Infants of actively immunized mothers acquire passive immunity that protects them from neonatal tetanus. Recovery from tetanus may not result in immunity; second attacks can occur and primary immunization is indicated after recovery.

1.15.9 Methods of control

1.15.9a Preventive measures

1. Educate the public on the necessity for complete immunization with tetanus toxoid, the hazards of puncture wounds and closed injuries that are particularly liable to be complicated by tetanus, and the potential need after injury for active and/or passive prophylaxis.
2. Universal active immunization with adsorbed tetanus toxoid (TT), which gives durable protection for at least 10 years; after the initial basic series has been
completed, single booster doses elicit high levels of immunity. In children under 7, the toxoid is generally administered together with diphtheria toxoid and pertussis vaccine as a triple (DTP or DTaP) antigen, or as double (DT) antigen when contraindications to pertussis vaccine exist.

3. Prophylaxis in wound management: Tetanus prophylaxis in patients with wounds is based on careful assessment of whether the wound is clean or contaminated, the immunization status of the patient, proper use of tetanus toxoid and/or TIG, wound cleaning and, where required, surgical debridement and the proper use of antibiotics.

1.15.9b Control measures
1. Report to local health authority.
2. Isolation: Not applicable.
3. Concurrent disinfection: Not applicable.
4. Quarantine: Not applicable.
5. Immunization of contacts: Not applicable.
6. Investigation of contacts and source of infection: Case investigation to determine circumstances of injury.
7. Specific treatment: TIG IM in doses of 3000–6000 IU. If immunoglobulin not available, tetanus antitoxin (equine origin) in a single large dose should be given IV following appropriate testing for hypersensitivity. Metronidazole, the most appropriate antibiotic in terms of recovery time and case-fatality, should be given for 7–14 days in large doses; this also allows for a reduction in the amount of muscle relaxants and sedatives required. The wound should be debrided widely and excised if possible. Wide debridement of the umbilical stump in neonates is not indicated. Maintain an adequate airway and employ sedation as indicated; muscle relaxant drugs together with tracheostomy or nasotracheal intubation and mechanically assisted respiration may be lifesaving. Active immunization should be initiated concurrently with treatment.

1.15.10 Management of the disease

- **Neonatal tetanus**
  - Disinfection of umbilical cord
  - Sedation
  - Anti-tetanus serum and antibiotics
  - Breast-milk through naso-gastric tube
- **Wound-related tetanus**
  - Adequate wound disinfection and debridement
  - Sedation
  - Anti-tetanus serum and antibiotics

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An infant suffering from neonatal tetanus

Centers for Disease Control and Prevention’s Public Health Image Library
1.16 Food Poisoning

ICD-10 A02 & A05

1.16.1 Identification

It is a frequent and inaccurate term of food intoxication which is an illness acquired through consumption of contaminated food. Outbreaks due to food consumption are suspected when an illness of short incubation period occurs after the consumption of contaminated food. It could be caused by:

1. Chemical contaminants such as heavy metals and organic compounds or
2. The more frequent causes of foodborne illnesses are:
   a) Toxins elaborated by bacterial growth in the food before consumption (Clostridium botulinum, Staphylococcus aureus, and Bacillus cereus; scombroid fish poisoning- associated not with a specific toxin but with elevated histamine levels) or in the intestines (Clostridium perfringes).
   b) Bacterial, viral, or parasitic infections (Brucellosis, Campylobacter enteritis, diarrhea caused by Escherichia coli, hepatitis A, listeriosis, trichinosis, and infection with Vibrio).
   c) Toxins produced harmful algal species or present in specific species (puffer fish poisoning, AZP).

Food borne disease is recognized by the occurrence of illness within short period (few hours to few weeks) after a meal.

Case classification

Suspect case: sudden onset (duration according to causes mentioned above) with severe nausea and vomiting, diarrhea, fever, fatigue, seizure, abdominal cramps, dry mouth, difficulty in swallowing and speaking, lowered blood pressure, blurred vision, and/or bloody stool.

Confirmed case: isolation of causative agent from suspected food, vomitus or stool (Staph. aureus); identification of bacteria in a culture from the suspected food or patient stool (Clost. perfringens); enterotoxin detection in the suspected food; suspect case among food poisoning cases sharing the same food, with at least one lab confirmed case.

NOTE: infectious agent, occurrence, reservoir, mode of transmission, incubation period, period of communicability, susceptibility and resistance will all depend on the exact causative agent. General methods of control are discussed below.

1.16.9 Methods of control

Any suspected food poisoning cases should be reported immediately to health authority and a case investigation should be conducted for all suspect and confirmed cases. Samples of stool, vomit, and suspected food should be collected. Instructions to contacts: not to eat suspected food.
1.16.10 Management of the disease

Because most cases of acute gastroenteritis are self-limited, specific treatment is not necessary. Some studies have quantified that only 10% of cases require antibiotic therapy.

- The main objective is adequate rehydration and electrolyte supplementation. This can be achieved with either an oral rehydration solution (ORS) or intravenous solutions (e.g., isotonic sodium chloride solution, lactated Ringer solution). Strict personal hygiene should be practiced during the illness.
  - Oral rehydration is achieved by administering clear liquids and sodium-containing and glucose-containing solutions. A simple ORS may be composed of 1 level teaspoon of salt and 4 heaping teaspoons of sugar added to 1 liter of water.
  - The use of ORS has reduced the mortality rate associated with cholera from higher than 50% to less than 1%.
  - ORS also is indicated in other dehydrating diarrheal diseases.
  - ORS promotes cotransport of glucose, sodium, and water across the gut epithelium, a mechanism unaffected in cholera.
  - The World Health Organization (WHO) recommends a solution containing 3.5 g of sodium chloride, 2.5 g of sodium bicarbonate, 1.5 g of potassium chloride, and 20 g of glucose per liter of water.
- Intravenous solutions are indicated in patients who are severely dehydrated or who have intractable vomiting.

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Group 2: Weekly Reported Diseases
2.1 Acute Diarrhea

ICD-10 A09

2.1.1 Identification

It is defined as passage of loose or watery stools at an increased frequency from normal (as 3 or more loose or watery stools per 24 hours). It is associated with other systemic or gastrointestinal symptoms including vomiting, fever, dehydration and electrolyte disturbances.

From practical clinical point, diarrheal illnesses can be divided into 3 clinical presentations:

1. Acute watery diarrhea (including cholera), lasting less than 14 days.
2. Acute bloody diarrhea (dysentery), lasting less than 14 days. Bloody diarrhea is that when the loose or watery stool contains visible red blood.
3. Persistent diarrhea, lasting 14 days or longer.

2.1.2 Infectious agent

Acute diarrhea may be caused by different bacterial (cholera, shigella, salmonella, E coli, Yersinia), viral (Rotavirus) and parasitic (giardia) enteric agents. The most common pathogens for acute watery diarrhea:

1. Rotavirus
2. ETEC (Enterotoxigenic Escherichia coli)
3. *Vibrio cholerae* - most important cause of epidemic watery diarrhea in developing countries due to high rate of morbidity and mortality. ETEC can also cause epidemic diarrhea, but its treatment is essentially the same as cholera.

NOTE: Occurrence, Reservoir, Mode of transmission, Incubation period, period of communicability, susceptibility and resistance, and methods of control all depend on the causative agent.

2.1.10 Management of the disease

The main objective is adequate rehydration and electrolyte supplementation. This can be achieved with either an oral rehydration solution (ORS) or intravenous solutions (e.g., isotonic sodium chloride solution, lactated Ringer solution). Strict personal hygiene should be practiced during the illness.

- Oral rehydration is achieved by administering clear liquids and sodium-containing and glucose-containing solutions. A simple ORS may be composed of 1 level teaspoon of salt and 4 heaping teaspoons of sugar added to 1 liter of water.
- The use of ORS has reduced the mortality rate associated with cholera from higher than 50% to less than 1%.
- ORS also is indicated in other dehydrating diarrheal diseases.

ORS promotes cotransport of glucose, sodium, and water across the gut epithelium, a mechanism unaffected in cholera.

The World Health Organization (WHO) recommends a solution containing 3.5 g of sodium chloride, 2.5 g of sodium bicarbonate, 1.5 g of potassium chloride, and 20 g of glucose per liter of water.

(A) Lupoid leishmaniasis with scarring due to previous *L. tropica* infection and the presence of red/brown papules at the periphery of the scar. (B) Lupoid leishmaniasis with scarring due to previous *L. tropica* infection, and two red/brown nodules at the superior and inferior poles, of the scar on the left cheek of a 9 year old boy from Afghanistan

44 http://adc.bmj.com/content/90/5/530/F3.large.jpg
2.2 Cutaneous Leishmaniasis

ICD-10 B55.1

2.2.1 Identification

A protozoan disease of skin and mucous membranes caused by species of the genus *Leishmania*. Single or multiple skin lesions starts with a papule that enlarges and typically becomes an indolent ulcer (wet ulcer with bad odor in *L. major* or dry type ulcer with smaller size in *L. tropica*). Lesions may heal spontaneously within weeks to months or last for a year or more leaving permanent scars on skin. Multiple ulcers following scarring may lead to skin deformity. Areas affected are mostly face and upper arms (uncovered areas in the body). The healing may last 3 – 6 months and may be more, leaving immunity for many years.

Affects all ages and sexes equally.

Laboratory criteria for diagnosis
– positive parasitology (stained smear or culture from the lesion)
– mucocutaneous leishmaniasis only: positive serology (IFA, ELISA).

**Case classification**

WHO operational definition:
A case of cutaneous leishmaniasis is a person showing clinical signs (skin or mucosal lesions) with parasitological confirmation of the diagnosis (positive smear or culture) and/or, for mucocutaneous leishmaniasis only, serological diagnosis.

2.2.2 Infectious agent

1. *Leishmania tropica* parasite
2. *Leishmania major* parasite

2.2.3 Occurrence

**Cases of Cutaneous Leishmaniasis, 2000-2009**

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Cases are reported in all provinces except Erbil, Dahuk.
The cases registered mostly in Maysan, Diyala, Salahedin.

2.2.4 Reservoir

Dogs, rodents, jackals, and humans.
2.2.5 Mode of transmission

From the reservoir host by the bite of infected female sand fly. Female sandflies pick up parasite (amastigote or LD bodies) while feeding on an infected host. Parasite undergo morphological change to become flagellate, development and multiplication in the gut of sandflies and move to mouthparts.

2.2.6 Incubation period

From one month or less; may be up to one month to one year.

2.2.7 Period of communicability

Sand fly activity occurs in humid conditions when there is no rain or wind. They are usually most active at dawn, dusk and during the night, or if they are disturbed in their hiding places (animal burrows, holes in houses and other relatively cool, humid locations) in the daytime.

2.2.8 Susceptibility and resistance

Geographical distribution of leishmaniasis is limited by the distribution of the sand fly, its susceptibility to cold climates, its tendency to take blood from humans or animals. Risk factor is the movement of susceptible populations into endemic areas, including large-scale migration of populations for economic reasons. Poverty and malnutrition play a major role in the increased susceptibility. Immunosuppression due to HIV predisposes to progression to visceral form.

2.2.9 Methods of control

1. Case detection and rapid treatment from hospital and dermatological department weekly and monthly.
2. Two round spraying yearly by insecticide with residual action to control vector (sand fly) in the affected areas.
3. Fogging by especially machine at evening in the affected areas.
4. Rodent control measures.
5. Stray dogs control measures.
6. Specific treatment mainly with pentavellant antimony compounds (pentostam) either by local infiltration around the lesion or systemic route in multiple lesions as follows (20 mg/kg/day) for 2 weeks.

2.2.10 Management of the disease

- Medicines called antimony-containing compounds are the main drugs used to treat leishmaniasis. These include:
  - Meglumine antimoniate
  - Sodium stibogluconate
- Other drugs that may be used include:
  - Amphotericin B

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Communicable Disease Control Guidelines

- Ketoconazole
- Miltefosine
- Paromomycin
- Pentamidine

- Cure rates are high with the proper medicine. Patients should get treated before damage to the immune system occurs. Cutaneous leishmaniasis may lead to disfigurement.

(A) Lupoid leishmaniasis with scarring due to previous *L. tropica* infection and the presence of red/brown papules at the periphery of the scar. (B) Lupoid leishmaniasis with scarring due to previous *L. tropica* infection, and two red/brown nodules at the superior and inferior poles, of the scar on the left cheek of a 9 year old boy from Afghanistan

47 http://adc.bmj.com/content/90/5/530/F3.large.jpg
2.3 Visceral Leishmaniasis

ICD-10 B55.0

2.3.1 Identification

Disease affects usually younger ages (below 5 years) in both sexes. The patients most commonly are from rural affected areas.

Death rate usually is 10 – 30% and may be more in advanced cases and especially without or delay treatment.

**Clinical description**

Clinical: systemic disease characterizes by gradual or sudden onsite fever which is persistent and regular. Other associate features include: hepatosplenomegaly, lymphadenopathy, anemia, leucopenia, thrombocytopenia and progressive emaciation and general weakness, brownish coloration of skin with progressive enlargement of abdomen.

Lab diagnosis: direct smear from the bone marrow (to find parasite amastigote), with bone marrow smear culture 3N media to find promastigote—with serological test (IFAT-ELISA-Rk39 dipstick).

2.3.2 Infectious agent

Intercellular protozoa of the genus *Leishmania* (*L. donovani* or *L. infantum*)

2.3.3 Occurrence

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Cases are reported in all provinces except Erbil, Dahuk, and Sulaymaniya. The cases registered mostly in Babil, Diyala, and Basrah.

The disease increase usually in autumn and winter every year and prevalent in the middle and south provinces of Iraq.

2.3.4 Reservoir

Dogs, Rodents, Jackals, Foxes

2.3.5 Modes of transmission

From the reservoir host by the bite of infected female sand fly.
Female sandflies pick up parasite (amastigote or LD bodies) while feeding on an infected host. Parasite undergo morphological change to become flagellate, development and multiplication in the gut of sandflies and move to mouthparts.

2.3.6 Incubation period

Usually from 1-3 months. (Range: 2 week-one year).

2.3.7 Period of communicability

Sand fly activity occurs in humid conditions when there is no rain or wind. They are usually most active at dawn, dusk and during the night, or if they are disturbed in their hiding places (animal burrows, holes in houses and other relatively cool, humid locations) in the daytime.

2.3.8 Susceptibility and resistance

Geographical distribution of leishmaniasis is limited by the distribution of the sand fly, its susceptibility to cold climates, its tendency to take blood from humans or animals. Risk factor is the movement of susceptible populations into endemic areas, including large-scale migration of populations for economic reasons. Poverty and malnutrition play a major role in the increased susceptibility.

2.3.9 Methods of control

1. Case detection and rapid treatment from hospital and dermatological department weekly and monthly.
2. Two round spraying yearly by insecticide with residual action to control vector (sand fly) in the affected areas.
3. Fogging by especially machine at evening in the affected areas.
4. Rodent control measures.
5. Stray dogs control measures.
6. Specific treatment mainly by pentavellant antimony compounds (pentostam) by systemic route as follows (20 mg/ kg/ day) for 28 days.

2.3.10 Management of the disease

- Medicines called antimony-containing compounds are the main drugs used to treat leishmaniasis. These include:
  - Meglumine antimoniate
  - Sodium stibogluconate
- Other drugs that may be used include:
  - Amphotericin B

Ketoconazole
Miltefosine
Paromomycin
Pentamidine

Cure rates are high with the proper medicine. Patients should get treated before damage to the immune system occurs.
Group 3: Monthly Reported Diseases
3.1 Viral Hepatitis

ICD-10 B15: Acute Hepatitis A
ICD-10 B16: Acute Hepatitis B
ICD-10 B17: Other Acute Viral Hepatitis
ICD-10 B18: Chronic Viral Hepatitis
ICD-10 B19: Unspecified Viral Hepatitis

3.1.1 Identification

1) Hepatitis Types A and E

Clinical features
These share same clinical course; no evidence of chronic form. Infection occurs in childhood and young adults asymptotically or with a mild illness- may be detectable only through laboratory tests of liver function.

Onset of illness in adults is usually abrupt with fever, malaise, anorexia, nausea and abdominal discomfort, followed within a few days by jaundice. The disease varies in clinical severity from a mild illness lasting 1–2 weeks to a severely disabling disease lasting several months. Prolonged, relapsing hepatitis for up to 1 year occurs in 15% of cases. Convalescence is often prolonged. In general, severity increases with age, but complete recovery without sequelaes or recurrences is the rule. Case-fatality for Hepatitis A is normally low, 0.1%–0.3%; it can reach 2.7% for adults over 50; persons with chronic liver disease have an elevated risk of death from fulminant hepatitis A. The case-fatality rate for HEV is similar to that of hepatitis A except in pregnant women, where it may reach 20% among those infected during the third trimester of pregnancy.

Laboratory Diagnosis of types A and E:
Acute Hepatitis A: demonstration of serum IgM anti-HAV detectable 5–10 days after exposure.
Acute hepatitis E: presence of IgM anti-HEV or exclusion of other causes of hepatitis, especially hepatitis A, by serological means.

2) Hepatitis Types B, and C

Clinical features
Usually asymptomatic. In those with clinical illness, the onset is usually insidious, with anorexia, vague abdominal discomfort, nausea and vomiting, some- times arthralgia and rash, often progressing to jaundice. Fever may be absent or mild. Less than 10% of children and 30%–50% of adults with acute hepatitis B virus (HBV) infection show icteric disease.

Severity ranges from unapparent cases detectable only by liver function tests to fulminating, fatal cases of acute hepatic necrosis. The case-fatality rate is about 1%; higher in those over 40. Fulminant HBV infection also occurs in pregnancy and among newborns of infected mothers. The risk of developing chronic infection varies inversely with age; occurs among about 90% of infants infected at birth, 20%–50% of children infected from 1 to 5 years, and 1%–10% of persons infected as older children and adults. Persons with chronic infection may or may not have a history of clinical hepatitis. About one-third have elevated aminotransferases; biopsy findings range from normal to chronic active hepatitis, with or without cirrhosis. An estimated
15%–25% of persons with chronic HBV infection will die prematurely of either cirrhosis or hepatocellular carcinoma.

Onset of acute hepatitis C is usually insidious, with anorexia, vague abdominal discomfort, nausea and vomiting; progression to jaundice less frequent than with hepatitis B. Asymptomatic in more than 90% of cases or mild, a high percentage (50%–80%) develop a chronic infection. Of chronically infected persons, about half will eventually develop cirrhosis or cancer of the liver.

Type D: The delta agent is a defective virus. It occurs in 2 forms, either coinfection or superinfection with hepatitis B virus. It is prevented through the prevention of Hepatitis B.

**Laboratory Diagnosis of types B and C:**
- **Type B:** serum HBsAg positive from several weeks before onset of symptoms to days, weeks or months after onset; it persists in chronic infections. The presence of HBsAg indicates that the person is potentially infectious. High titers of IgM anti-HBc occur during acute infection—IgM anti-HBc usually disappears within 6 months but can persist in some cases of chronic hepatitis; this test may reliably diagnose acute HBV infection. Anti HBCIgG replaces anti HBcIgM and continues forever.

- **Type C:** antibody to the hepatitis C virus (anti-HCV) - by the enzyme immunoassay (EIA) and the recombinant immunoblot assay. These tests do not distinguish between acute, chronic, or resolved infection. Acute or chronic HCV infection in a patient with a positive EIA test should be confirmed by a sensitive HCV RNA assay. Quantitative determination of HCV levels provides information on the likelihood of response to treatment in patients undergoing antiviral therapy. Liver biopsy can provide direct histological assessment of liver injury due to HCV but cannot be used to diagnose HCV infection. Genotyping of HCV infection is important from epidemiological and treatment point of view.

**Case definition**
An acute illness that includes malaise, extreme fatigue, fever, nausea and sometimes vomiting and upper right quadrant abdominal tenderness, then dark urine followed by jaundice.

**Case classification**
- **Suspected case:** A case compatible with clinical description.
- **Probable case:** A Suspected Case + Positive Bile pigment in urine and elevated serum bilirubin and liver enzymes (ALT, SGPT and Serum Alkaline Phosphatase).
- **Confirmed case:** Probable/suspected case with positive specific serological tests. This is most commonly done by detecting Anti-HAV, HBsAg, Anti-HCV, and Anti-HEV. This can be done by different methods and the most famous one is ELISA method. In addition great increase of liver enzymes like alanine transaminase (ALT), serum alkaline phosphatase and SGOT etc.

**Chronic Hepatitis B cases:** Any patient with positive HBsAg for more than 6 months and is considered as a case of chronic carrier state.

Note: The patient should be tested for other markers (HBeAg, Anti- HBCIgM, and Anti- HBe) to determine the health status and infectivity level. Positivity for HBeAg indicates high infectivity while positivity for Anti- HBCIgM means acute infection. Positive Anti- HBe means less Infectivity.
**Chronic Hepatitis C**: Any patient who is positive for HCV Abs should be referred to the specialist center for more evaluation because positivity for HCV Abs cannot differentiate infection from immunity and needs further investigations. PCR is very necessary to identify such cases.

**3.1.2 Infectious agent**

**Hepatitis A virus (HAV)**: RNA virus, family *Picornaviridae*.

**Hepatitis B virus (HBV)**: a *hepadnavirus*, partially double-stranded DNA virus composed of nucleocapsid core (HBcAg), surrounded by an outer lipoprotein coat containing the surface antigen (HBsAg). 8 main genotypes (A-H).

**Hepatitis C virus (HCV)**: RNA virus, genus *Hepacivirus*, *Flaviviridae* family. At least 6 genotypes and approximately 100 subtypes.

**Hepatitis E virus (HEV)**: a spherical, nonenveloped, single-stranded RNA virus, family *Hepeviridae*.

**3.1.3 Occurrence**

**Type A**: Iraq is considered highly endemic as indicated by 96.4% prevalence of Anti HAV Abs.

**Type B**: Iraq is considered with low endemic with HBsAg prevalence was 1.6% in 2006.

**Type C**: In the Middle East, the prevalence of anti-HCV ranges from 1% to more than 12%. In Iraq, the prevalence of anti HCV Abs was found to be 0.4%.

**Type E**: The prevalence of Anti HEV Abs in Iraq in 2006 was about 20%.

**Rate of occurrence for various types of viral hepatitis in Iraq from 2005 to 2010 can be summarized in the following table:**

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<th>year</th>
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<th>Occurrence rate (per 100000)</th>
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<tr>
<td>2010</td>
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</tbody>
</table>

**3.1.4 Reservoir**

**Type A**: Humans, rarely chimpanzees and other primates.

**Types B and C**: Humans. An animal reservoir in nature has not been recognized.

**Type E**: Man is the natural host; some non-human primates, e.g. chimpanzees. Natural infections have been described in pigs, chicken and cattle, particularly in highly endemic areas.

**3.1.5 Mode of transmission**

**Types A and E**: Person-to-person by the fecal-oral route. Common source outbreaks related to contaminated water; food contaminated by infected food handlers, including foods not cooked or
handled after cooking; raw or undercooked mollusks harvested from contaminated waters; and contaminated produce such as lettuce and strawberries. Hepatitis E may in fact be a zoonotic infection with coincident areas of high human infection.

**Types B and C:** Transmission occurs by percutaneous (IV, IM, SC, intradermal) and permucosal exposure to infective body fluids. Because HBV is stable on environmental surfaces for at least 7 days, indirect inoculation of HBV can occur via inanimate objects. Fecal-oral or vector-borne transmission has not been demonstrated. Major modes of HBV transmission include sexual or close household contact with an infected person, perinatal mother-to-infant transmission, injecting drug use and nosocomial exposure. Transmission of HBV in households primarily occurs from child to child. Communally used razors and toothbrushes have been implicated as occasional vehicles of HBV transmission in this setting. Perinatal transmission is common, especially when HBV-infected mothers are also HBeAg-positive.

HCV is primarily transmitted parenterally. Sexual and mother-to-child have been documented but appears far less efficient or frequent than the parenteral route.

### 3.1.6 Incubation period

**Type A:** Average 28–30 days (range 15–50 days).  
**Type B:** Usually 45–180 days, average 60–90 days.  
**Type C:** Ranges from 2 weeks to 6 months; commonly 6–9 weeks.  
**Type E:** The range is 15 to 64 days; the mean varied from 26 to 42 days in various epidemics.

### 3.1.7 Period of communicability

**Type A:** maximum infectivity occurs during the latter half of incubation and continues for a few days after onset of jaundice (or during peak aminotransferase activity in anicteric cases). Most cases are probably noninfectious after the first week of jaundice.  
**Type B:** All persons who are HBsAg-positive are potentially infectious.  
**Type C:** From one or more weeks before onset of the first symptoms; may persist in most persons indefinitely. Peaks in virus concentration appear to correlate with peaks in ALT activity.  
**Type E:** Not known. HEV has been detected in stools 14 days after the onset of jaundice and approximately 4 weeks after oral ingestion of contaminated food or water; it persists for about 2 weeks.

### 3.1.8 Susceptibility and resistance

For all types, susceptibility is general. Immunity against HBV is believed to persist for at least 15 years after successful immunization.

### 3.1.9 Methods of control

#### 3.1.9a Preventive measures

**Types A and E:**
1. Educate the public about good sanitation and personal hygiene: hand washing and safe disposal of feces.
2. Provide proper water treatment and distribution systems and sewage disposal.
3. Hepatitis A vaccine is recommended in high risk groups. High-risk groups include the following:
   i. People with chronic liver disease or clotting factor disorders, men who have sex with men, injecting drug users, handling HAV in research laboratory settings;
   ii. In outbreak situations: community and the feasibility of rapidly implementing a widespread vaccination program.
4. Oysters, clams and other shellfish from contaminated areas should be heated to a temperature of 85°–90°C (185°–194°F) for 4 minutes or steamed for 90 seconds before eating. In endemic areas, travelers should take only hot or bottled beverages and hot, well-cooked food.

**Type B:**

1. Vaccination
   a) Hepatitis B vaccine: anti-HBs or anti-HBc testing is not required prior to immunization. Pregnancy is not a contraindication for receiving hepatitis B vaccine. Vaccine in Iraq is routinely given to newborn immediately after birth at the delivery room with subsequent doses at 2 months and 6 months of age. For infants born to HbsAg positive women, the schedule should be birth, 1–2 and 6 months of age. These infants should also receive 0.5 ml of HBIG.
   b) All household contacts who test negative for HbsAg should receive 3 doses of the vaccine.
   c) In addition, persons at high risk who should routinely receive pre-exposure hepatitis B immunization include: a) health care and public safety workers who are likely to handle blood or body fluids (midwives etc.); b) clients and staff of institutions for the developmentally disabled; c) hemodialysis patients; d) patients with bleeding disorders who receive blood products; e) high risk occupations (tattooists, hairdressers etc.).
2. a) Screening for a) all pregnant women for the presence of HbsAg. B) Preoperative patients for HbsAg. C) All foreigners who seek residency in Iraq and prevent giving them residency.
3. Use adequately sterilized syringes and needles (including acupuncture needles); use disposable equipment whenever possible. Discourage tattooing; enforce aseptic sanitary practices in tattoo parlors, including proper disposal of sharps.
4. In blood banks, all donated blood should be tested for HbsAg; reject donors with a history of viral hepatitis (positive HbsAg and Anti-HCV Ab), those who have a history of injecting drug use or show evidence of drug addiction or those who have received a blood transfusion or tattoo within the preceding 6 months.
5. Maintain surveillance for all cases of post transfusion hepatitis; keep a register of all people who donated blood for each case. Notify blood banks of potential carriers so that future donations may be identified promptly.
6. Nowadays, there is general agreement among public health authorities, that HBV-positive health care workers should not perform exposure-prone surgery or similar treatment of patients.
**Type C:** General control measures against HBV infection apply except for vaccination as there is no available vaccine for Hepatitis C.

### 3.1.9b Control measures

**Types A, B, C and E:**
1. Report (weekly and monthly) to local health authority.
2. **Isolation:** For **proven hepatitis A and E,** enteric precautions during the first 2 weeks of illness, but no more than 1 week after onset of jaundice; the exception is an outbreak in a neonatal intensive care setting, where prolonged enteric precautions must be considered.

**For Hepatitis B and C:** Universal precautions to prevent exposures to blood and body fluids.
3. **Concurrent disinfection:** For **Hepatitis A and E:** Sanitary disposal of feces, urine and blood.
   **For Hepatitis B and C:** disinfection of equipment contaminated with blood or infectious body fluids.
4. **Quarantine:** for A, B, C and E are not applicable.
5. **Immunization of contacts:**
   - **Type A:** Active immunization should be given as soon as possible, but no later than 2 weeks after exposure. Passive immunization with IG (IM), 0.02 ml/kg of body weight, should be given as soon as possible after exposure, but also within 2 weeks. Hepatitis A vaccine and IG are not indicated for contacts in the usual office, school or factory settings. IG should be administered to previously unimmunized persons in the situations listed below, preferably together with hepatitis A vaccine given concurrently at a separate injection site:
     1. Close personal contacts, including household, sexual, drug using and other close personal contacts.
     2. Attenders at day care centers if one or more cases of hepatitis A are recognized in children or employees or if cases are recognized in 2 or more households of attenders prophylaxis may be given to classroom contacts of an index case.
     3. In a common source outbreak, if a food handler is diagnosed with hepatitis A, hepatitis A vaccine and IG should be administered to other food handlers in the same establishment.
     4. Other unimmunized (not infected previously, not vaccinated) high risk groups e.g. blood disorders that require frequent blood transfusions, Hepatitis B and C patients, renal dialysis patients, immunocompromised patients, etc.
   - **Type B:** Products available for post exposure prophylaxis include HBIG and hepatitis B vaccine. When indicated, administer HBIG as soon as possible after exposure in the following situation:
     a) Infants born to HBsAg positive mothers should receive a single dose of vaccine within 12 hours of birth and HBIG (0.5 ml IM), the first dose of vaccine to be given concurrently with HBIG but at a separate site; second and third doses of vaccine (without HBIG) 2 and 6 months later.
b) After percutaneous (e.g. needle stick) or mucous membrane exposures to blood that might contain HBsAg, a decision to provide post exposure prophylaxis must include consideration of: i) whether the source of the blood is available; ii) the HBsAg status of the source; iii) the hepatitis B immunization status of the exposed person.

For previously unimmunized persons exposed to blood from an HBsAg positive source, a single dose of HBIG (0.06 ml/kg, or 5 ml for adults) should be given as soon as possible, but at least within 24 hours of high-risk needle-stick exposure, and the hepatitis B vaccine series should be started. If active immunization cannot be given, another dose of HBIG should be given 1 month after the first. HBIG is not usually given for needle stick exposure to blood that is not known or highly suspected to be positive for HBsAg, since the risk of infection in these instances is small; however, initiation of hepatitis B immunization is recommended if the person has not previously been immunized.

For previously immunized persons exposed to an HBsAg positive source, post exposure prophylaxis is not needed in cases with a protective antibody response to immunization (anti-HBs titer of 10 milli-IUs/mL or greater). For persons whose response to immunization is unknown, hepatitis B vaccine and/or HBIG should be administered.

c) After sexual exposure to a person with acute HBV infection, a single dose of HBIG (0.06 ml/kg) is recommended if it can be given within 14 days of the last sexual contact. For all exposed sexual contacts of persons with acute and chronic HBV infection, vaccine should be administered.

- **Type C**: Available data indicate that post exposure prophylaxis with IG is not effective in preventing infection.
- **Type E**: No products are available to prevent hepatitis E.

6) Investigation of contacts and source of infection: For types A, B, C and E: Search for missed cases and maintain surveillance of contacts in the patient’s household or, in a common source outbreak, people exposed to the same risk.

7) Specific treatment:

- **Types A and E**: None.
- **Types B and C**: For acute cases: none. Alpha interferon, lamivudine and adefovir have been licensed for treatment of chronic hepatitis B. Candidates for therapy should have liver biopsy evidence of chronic hepatitis B; treatment is most effective in individuals in the high-replicative phase (HBeAg positive) of infection because they are the most likely to be symptomatic, infectious and at risk of long-term sequelae. Approximately 10% of patients who respond lose HBsAg 6 months after therapy. Lamivudine has fewer side-effects and is easier to administer, but has a modest efficacy rate, requires long-term treatment to maintain response, and is associated with a high rate of viral resistance, particularly when prolonged. Adefovir is an antiviral drug active against both wild-
Communicable Disease Control Guidelines

type and lamivudine-resistant HBV, and has an antiviral activity similar to that of lamivudine. For the treatment of chronic hepatitis C, highest response rates (40–80%) have been achieved with a combination therapy of ribavirin and slow-release interferons (“pegylated interferons”), making it the treatment of choice. Determination of genotype influences treatment decisions. However, these medications have significant side-effects that require careful monitoring. Ribavirin is a teratogen; thus pregnancy should be avoided during treatment. Corticosteroids and acyclovir have not been effective.

3.1.9c Epidemic measures
- **Types A and E:**
  - Identify the population exposed. Eliminate common sources of infection.
  - Effective use of hepatitis A vaccine in community-wide outbreak situations requires the identification of an appropriate target group for immunization, the initiation of immunization early in the course of the outbreak and the rapid achievement of high (approximately 70% at least) first-dose vaccine coverage levels. Specific outbreak control measures must be tailored to the characteristics of hepatitis A epidemiology and of the existing hepatitis A immunization program, if any, in the community.
  - Make special efforts to improve sanitary and hygienic practices to eliminate fecal contamination of foods and water.
  - Outbreaks in institutions may warrant mass prophylaxis with hepatitis A vaccine and IG.
- **Types B and C:** When 2 or more cases occur in association with some common exposure, search for additional cases. Institute strict aseptic techniques. If a plasma derivative such as antihemophilic factor, fibrinogen, pooled plasma or thrombin is implicated, withdraw the lot from use and trace all recipients of the same lot in a search for additional cases.

3.1.9d Disaster implications
- **Types A and E:** Hepatitis A is a potential problem in large collections of people with overcrowding, inadequate sanitation and water supplies; if cases occur, increased efforts should be exerted to improve sanitation and safety of water supplies. Mass administration of hepatitis A vaccine, which should be carefully planned, is not a substitute for environmental measures.
- **Types B and C:**
  Relaxation of sterilization precautions and emergency use of unscreened blood for transfusions may result in an increased number of cases.

E. International measures:
- **Types A and E:** None.
- **Types B and C:** Ensure adequate virus inactivation for all internationally traded biological products.
3.1.10 Management of the disease

Patient with suspected acute viral

Primary Health Care Center

TSB+bilirubin in urine

Positive

Case investigation form + serum (to be examined for all types

>=15years

HBsAg

Positive

Negative

Anti HCVAb

Positive

Negative

Anti HAV Ab

Positive

Negative

Anti HEV Ab

Positive

Negative

Non A,B,C,E

<15 years

Anti HAV IgM

Positive

Negative

Anti HEV Ab

Positive

Negative

HBsAg

Positive

Negative

Non A,B,C,E

Not a case of acute viral hepatitis unless positive for contact with a known case in close contacts.

50 Designed by the Communicable Disease Control Center, MOH, Iraq.
- **Hepatitis A:** There is no specific treatment for hepatitis A. Recovery from symptoms following infection may be slow and take several weeks or months. Therapy is aimed at maintaining comfort and adequate nutritional balance, including replacement of fluids that are lost from vomiting and diarrhea.\(^{51}\)

- **Hepatitis B:** There is no specific treatment for acute hepatitis B. Care is aimed at maintaining comfort and adequate nutritional balance, including replacement of fluids that are lost from vomiting and diarrhea. Chronic hepatitis B can be treated with drugs, including interferon and anti-viral agents, which can help some patients. Treatment can cost thousands of dollars per year and is not available to most patients in developing countries. Liver cancer is almost always fatal, and often develops in people at an age when they are most productive and have family responsibilities. In developing countries, most people with liver cancer die within months of diagnosis. In higher income countries, surgery and chemotherapy can prolong life for up to a few years in some patients. Patients with cirrhosis are sometimes given liver transplants, with varying success.\(^{52}\)

- **Hepatitis C:** Interferon and ribaviron-based therapy has been the mainstay of HCV treatment. Unfortunately, interferon is not widely available globally, is not always well tolerated, some genotypes respond better than others, and many people who take it do not finish their treatment. While HCV is generally considered to be a curable disease, for many persons this is not a reality. Fortunately, scientific advances and intense research and development have led to the development of many new oral antiviral drugs for HCV infection. The future seems to hold great promise for HCV specific oral drugs that will be more effective and better tolerated. Much still needs to be done to ensure that these advances lead to greater access and treatment globally.\(^{53}\)

- **Hepatitis E:** Hepatitis E is a viral disease, and as such, antibiotics are of no value in the treatment of the infection. There is no hyperimmune E globulin available for pre- or post-exposure prophylaxis. HEV infections are usually self-limited, and hospitalization is generally not required. No available therapy is capable of altering the course of acute infection. As no specific therapy is capable of altering the course of acute hepatitis E infection, prevention is the most effective approach against the disease. Hospitalization is required for fulminant hepatitis and should be considered for infected pregnant women.\(^{54}\)

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3.2 Brucellosis

ICD-10 A23

3.2.1 Identification

A systemic zoonotic bacterial disease caused by *Brucella* species.

**Clinical features**
An illness characterized by acute or insidious onset, with continued, intermittted or irregular fever of variable duration, profuse sweating particularly at night, fatigue, anorexia, weight loss, headache, arthralgia and generalized aching. Local infection of various organs may occur. Fever is the most common symptom and may be associated with a variety of other complaints. Osteo-articular complications are common (20 – 60% of cases).

**Case classification**
*Suspected*: A case that is compatible with the clinical description and is epidemiologically linked to suspected or contaminated animal products.
*Probable*: A suspected case that has a positive Rose Bengal test.
*Confirmed*: A suspected or probable case that at is laboratory-confirmed through isolation of Brucella spp. From clinical specimen or Brucella agglutination titer (e.g., standard tube agglutination tests: SAT>160 IU) in one or more serum specimens obtained after onset of symptoms or ELISA (IgA, IgG, IgM) 2-mercaptoethanol test, complement fixation test, (FAT), and radio immunoassay for counter-immunoelectrophoresis (CIEP).

3.2.2 Infectious agent

*B. abortus, B. melitensis, B. suis, and B. canis.*

3.2.3 Occurrence

Worldwide, especially in Mediterranean Countries of Europe, North and East Africa, and Central Asia. The organism and source of infection varies according to geographical area. Brucellosis used to be an occupational hazard in farm workers, veterinarians, and abattoir workers.

**Cases of brucellosis, 2000-2011**

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</tbody>
</table>

3.2.4 Reservoir

Infection is humans can be transmitted from cattle, goats, sheep, dogs, and swine.

3.2.5 Mode of transmission
• Contact with infected animal tissues, blood, urine, vaginal discharges, and aborted animal fetuses and especially placentae; also by ingestion of raw milk and dairy products from infected animals without boiling or pasteurizing milk used.

• Inhalation in laboratory workers.

3.2.6 Incubation period

Variable and difficult to ascertain. Usually 5 to 60 days; can be several months.

3.2.7 Period of communicability

There is no evidence of communicability from person to person.

3.2.8 Susceptibility and resistance

Severity and duration of clinical illness are subject to wide variation. Duration of acquired immunity is uncertain.

3.2.9 Methods of control

3.2.9a Preventive measures

• Education the public: Avoid drinking untreated, unpasteurized milk or eating dairy products from such milk. Boiling milk is effective when pasteurization is not available.

• Educate farmers and handlers of potentially infected animals to reduce exposure and exercise care in handling placentae, discharges and fetuses.

• Search for and investigate livestock at risk of infection.

3.2.9b Control measures

• Report to local health authority.

• Specific treatment: Doxycycline 100mg twice daily for 6 weeks.

• Inform the Department of Agriculture and municipality.

• Enquire into source of infection and trace infection to common source.

• Recall incriminated products. Stop distribution of milk and milk products unless pasteurization is instituted.

3.2.10 Management of the disease

• Usually, doxycycline and rifampin are used in combination for 6 weeks to prevent reoccurring infection. Depending on the timing of treatment and severity of illness, recovery may take a few weeks to several months. Mortality is low (<2%), and is usually associated with endocarditis.

3.3 Chicken pox

ICD-10 B01: Chicken Pox
ICD-10 B02: Herpes Zoster

3.3.1 Identification

Clinical features

Varicella (chickenpox): Chickenpox generally presents with a low-grade fever, malaise and a rash. The rash is firstly maculopapular then becomes vesicular (blistered) and progresses to crusted lesions over about five days. Lesions appear in three or four crops. They are most numerous on the trunk and less so on the face, scalp, limbs and mucous membranes of the mouth. Some cases (about 5%) are subclinical or exceedingly mild in nature. Adults tend to suffer with more severe disease than children. Rarely, the disease may be fatal. Complications include secondary bacterial infection of the skin lesions, primary varicella pneumonia, aseptic meningitis, encephalitis and Reye’s syndrome (acute encephalopathy with fatty infiltration and dysfunction of the liver). Newborns and immunosuppressed patients are at greatly increased risk of severe chickenpox.

Herpes zoster (shingles): Herpes zoster or shingles is characterized by a predominantly unilateral vesicular eruption within a dermatome. It is often associated with severe pain that may precede lesions by 48–72 hours. The rash lasts up to several weeks depending on severity. The rash is often more widespread and persistent in immunosuppressed patients. Patients must be carefully evaluated to ensure that there is no eye involvement when the rash involves the ophthalmic area of the face. Specialist treatment is mandatory in this case as blindness can result. Incidence increases with age and children under 12 are rarely affected unless immunosuppressed or infected as infants. A debilitating complication of herpes zoster in many (especially elderly) patients is prolonged pain (post-herpetic neuralgia) which may persist for months after resolution of the skin lesions.

Method of diagnosis

Confirmation of the diagnosis is generally only required when the clinical picture is atypical. It is made by:

- isolation of the virus in cell cultures
- visualization by electron microscopy
- serological tests for antibodies
- immunofluorescence on lesion swab or fluid
- nucleic acid testing or PCR.

3.3.2 Infectious agent

Human herpes virus 3 (alpha) or varicella zoster virus (VZV).
3.3.3 Occurrence

Chickenpox is a highly contagious but generally mild disease and is endemic in the population. It becomes epidemic among susceptible individuals mainly during winter and early spring. More than 90% of cases are children under 15 years of age. Herpes zoster (shingles) occurs in 20% of people, mostly when they are elderly due to the reactivation of latent virus from the dorsal root ganglia.

3.3.4 Reservoir

Humans are the only reservoir of the virus, and disease occurs only in humans.

3.3.5 Mode of transmission

Chickenpox transmission is mainly person to person by airborne respiratory droplets but also by direct contact with vesicle fluid of chickenpox cases, or contact with the vesicle fluid of patients with herpes zoster. Indirect contact occurs through articles freshly soiled by discharges from vesicles of infected persons. Scabs are not infective.

3.3.6 Incubation period

The incubation period is from two to three weeks, usually 14-16 days. This may be prolonged in immunosuppressed persons or following immunoglobulin administration as passive immunization against varicella.

3.3.7 Period of communicability

Usually communicable for one to two days (up to five days) before the onset of the rash, continuing until all lesions are crusted. Communicability may be prolonged in patients with altered immunity. Those with zoster are considered infectious for a week after lesions appear.

3.3.8 Susceptibility and resistance

Susceptibility to chickenpox is universal among those not previously infected. Over 80% of non-immune household contacts of a case of chickenpox will become infected. Patients who are at high risk of severe disease/complications if they do not have immunity include: infants less than one month old, pregnant women and immunosuppressed individuals including those with hematological malignancies, on chemotherapy, high dose steroids or with HIV infection.

3.3.9 Methods of control

3.3.9a Control measures

Communicable Disease Control Guidelines

- Symptomatic management of cases: Tepid bathing or cool compresses may help to alleviate itching.
- Children with chickenpox should be excluded for at least five days after the rash appears. A few remaining scabs are not a reason for continued exclusion. Children with shingles can attend school if the lesions can be covered adequately however exclusion from swimming and contact sports should be advised for seven days after the rash appears.
- Advise adults to stay away from work for the same period
- Avoid contact with high risk susceptible persons.
- Aspirin should never be given to children with varicella due to a strong association with the development of Reye’s syndrome.
- If chickenpox develops in pregnancy, refer within 24 hours of rash onset.

3.3.10 Management of the disease57

- Primary varicella infection in the healthy child is a rather benign disease that requires symptomatic therapy only. Oral acyclovir should be considered for healthy persons at increased risk of severe varicella infections.
- Adults and immunocompromised persons with chickenpox have a more complicated course than that occurring in children, and therefore, the condition necessitates a more aggressive pharmacotherapeutic approach. Intravenous acyclovir therapy is recommended for patients who are immunosuppressed or immunocompromised.
- Varicella-zoster immune globulin (VZIG) is indicated for use in highly susceptible, VZV-exposed immunocompromised or immunosuppressed populations.


3.4 Echinococcosis (Hydatid disease)

ICD-10 B67

3.4.1 Identification

A space occupying disease, in humans caused by the larval stage of the dog tapeworm, *Echinococcus granulosus*. The signs, symptoms and extent of disease are determined by the size and location of the unilocular cysts (hydatid cyst) that are characteristic of this disease. Symptoms develop as result of pressure, leakage and rupture of cysts. The most common site for the cysts is the liver; less commonly brain, lungs, kidney and uncommonly the heart, thyroid and bone. Cyst may remain viable or dies and calcifies. They may be detected on routine x-ray.

**Case classification**

*Suspected*: clinically suspected case with history of contact to animals or animal viscera
*Probable*: diagnosis by plain x-ray, ultrasound or CT, casoni test
*Confirmed*: Laboratory (PCR, IFAT, ELISA)

3.4.2 Infectious agent

*Echinococcus granulosus*

3.4.3 Occurrence

Cases of hydatid disease, 2000-2011

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</table>

3.4.4 Reservoir

Dogs and foxes. Major intermediate hosts include goats, sheep, and cattle.

3.4.5 Mode of transmission

Infection occurs by hand-to mouth transfer of tapeworm eggs from dog faces. The larvae penetrate the intestinal mucosa, enter the portal system and carried to various organs where they produce the cysts. The important life cycle is the dog-sheep-dog.

3.4.6 Incubation period
Communicable Disease Control Guidelines

Variable, from months to years.

3.4.7 Period of communicability

Not communicable through person-to-person transmission. Dogs pass eggs approximately seven weeks after infection. In the absence of reinfection, this ends in about one year.

3.4.8 Susceptibility and resistance

Adults (ages 15-49) are more often infected than children.

3.4.9 Methods of control

3.4.9a Preventive measures

- Educate the public on the danger of close association with dogs and on the need to wash hands after contact with dogs.
- Treat infected dogs and destroy unwanted dogs.
- Control slaughter of animals, particularly sheep. The area should be enclosed to prevent the access by dogs and have adequate drainage, an incinerator and/or disposal pit.

3.4.9b Control measures

- Monthly report of cases to health authority.
- No need for isolation, or concurrent disinfection.
- Surgery is treatment of choice. Mebendazole is used when surgery is contraindicated.
- Household contacts should be examined for evidence of infection.
- Dogs on contact with patient should be investigated.

3.4.10 Management of the disease58

- Two benzimidazolic drugs, mebendazole and albendazole, are the only anthelmintics effective against cystic echinococcosis. Albendazole and mebendazole are well tolerated but show different efficacy.
  - Albendazole is significantly more effective than mebendazole in the treatment of liver cysts. Benzimidazole treatment alone requires prolonged administration over many weeks, with an unpredictable outcome in terms of response rates in individuals.
  - Treatment with albendazole in E granulosus infection can result in an apparent cure in as many as 30% of patients, with a further 40-50% of patients showing objective evidence of response when observed short term. Patients who do not

show obvious initial evidence of response may be found to be cured when observed over several years.

- Albendazole efficacy increases with courses of up to 3 months in the more common cyst sites. Overall, albendazole has been demonstrated to be a useful advance in the management of cystic echinococcosis when used as sole treatment or as an adjunct to surgery or other treatments.

Appearance of a typical cyst at removal

3.5 Leprosy

ICD-10 A30

3.5.1 Identification

A chronic bacterial disease of the skin, peripheral nerves and (in lepromatous patients) the upper airway. The clinical manifestations of the disease vary in a continuous spectrum between 2 polar forms: i) lepromatous (multibacillary) leprosy: symmetrical and bilateral nodules, papules, macules and diffuse infiltrations, usually numerous and extensive; involvement of the nasal mucosa may lead to crusting, obstructed breathing and epistaxis; ocular involvement leads to iritis and keratitis; ii) tuberculoid (paucibacillary) leprosy: skin lesions single or few, sharply demarcated, anaesthetic or hypoaesthetic; bilateral asymmetrical involvement of peripheral nerves tends to be severe. Borderline leprosy has features of both polar forms and is more labile. Indeterminate leprosy is characterized by hypo pigmented maculae with ill-defined borders; if untreated, it may progress to tuberculoid, borderline or lepromatous disease.

Case definition

A case of leprosy is a person having one or more of the following, who has yet to complete a full course of treatment:

- Hypo pigmented or reddish skin lesion(s) with definite loss of sensation
- Involvement of the peripheral nerves (definite thickening with loss of sensation)
- Skin smear positive for acid-fast bacilli.
- The operational case definition includes retrieved defaulters with signs of active disease and relapsed cases who have previously completed a full course of treatment. It does not include cured persons with late reactions or residual disabilities.

Clinical diagnosis is based on complete skin examination. Search for signs of peripheral nerve involvement (hyperesthesia, anesthesia, paralysis, muscle wasting or trophic ulcers) with bilateral palpation of peripheral nerves (ulnar nerve at the elbow, peroneal nerve at the head of the fibula and the great auricular nerve) for enlargement and tenderness. Test skin lesions for sensation (light touch, pinprick, temperature discrimination). The clinical manifestations can include “reactions” of leprosy, i.e. acute adverse episodes, which are termed erythema nodosum leprosum in lepromatous patients and reversal reactions in borderline leprosy. Differential diagnosis includes many infiltrative skin diseases, including lymphomas, lupus erythematosus, psoriasis, scleroderma and neurofibromatosis. Diffuse cutaneous leishmaniasis, some mycoses, myxoedema and pachydermoperiostosis may resemble lepromatous leprosy, but acid-fast bacilli are not present. Several skin conditions, such as vitiligo, tinea versicolor, pityriasis alba, nutritional dyschromia, nevus and scars may resemble tuberculoid leprosy.

Laboratory criteria include the presence of alcohol-acid-fast bacilli in skin smears (scrape-incision method). In the paucibacillary form the bacilli may be so few that they are not demonstrable. In view of the increasing prevalence of HIV and hepatitis B infection in many countries where leprosy remains endemic, the number of skin smear sites and the frequency of
smear collection should be limited to the minimum necessary. In practice, laboratories are not essential for the diagnosis of leprosy.

Leprosy cases can be classified as follows:
- Multibacillary leprosy: more than 5 patches or lesions on the skin
- Paucibacillary leprosy: 1 to 5 patches or lesions on the skin.

3.5.2 Infectious agent

*Mycobacterium leprae.* This cannot be grown in bacteriological media or cell cultures.

3.5.3 Occurrence

During 2002, 620 000 persons were diagnosed with leprosy, 90% of them in Brazil, India, Madagascar, Mozambique, Nepal, and in the United Republic of Tanzania. Control has improved with the introduction of multidrug therapy (MDT). WHO has targeted the disease for elimination (less than 1 case/10 000 population) and this has been achieved in 110 out of the 122 countries endemic in 1985. Newly recognized cases in the USA are few and diagnosed principally in California, Florida, Hawaii, Louisiana, Texas and in New York City, and in Puerto Rico. Most of these cases are in immigrants and refugees whose disease was acquired in their native countries; however, the disease remains endemic in California, Hawaii, Louisiana, Texas and Puerto Rico.

3.5.4 Reservoir

Humans are the only known significant reservoirs. There have been reports that disease in armadillos has been naturally transmitted to humans. Naturally acquired leprosy has been observed in a mangabey monkey and in a chimpanzee captured in Nigeria and Sierra Leone, respectively.

3.5.5 Mode of transmission

The mode of transmission is not clearly established. The disease is in all likelihood transmitted from the nasal mucosa of a patient to the skin and respiratory tract of another person. Transmission requires close contact. Although the bacillus can survive up to 7 days in dried nasal secretions, indirect transmission is unlikely.

3.5.6 Incubation period

Ranges from 9 months to 20 years, the average is probably 4 years for tuberculoid leprosy and twice that for lepromatous leprosy
3.5.7 Period of communicability

Leprosy is not usually infectious after three months of continuous treatment with dapsone or clofazimine, or after two to three weeks of treatment with rifampicin.

3.5.8 Susceptibility and resistance

Everyone is susceptible to infection. Household and prolonged close contact especially with a multi-bacillary case seems important. Children aged between five and nine years are at greatest risk. The disease is rarely seen in children under age 3. The risk of progression to leprosy disease following infection is considered to be approximately the same as tuberculosis which is approximately a 10% lifetime risk.

3.5.9 Methods of control

The availability of effective and time-limited ambulatory treatment, with rapid elimination of infectiousness, has changed management. Hospitalization should now be limited only to cases such as the surgical correction of deformities, treatment of ulcers resulting from anesthesia, and severe leprosy reactions.

3.5.9a Preventive measures

- Early detection and treatment of cases.
- Dapsone chemoprophylaxis is not recommended (limited effectiveness and danger of resistance).
- Health education together with counseling of patients and relatives must stress the availability of effective multidrug therapy, the absence of infectivity of patients under continuous treatment and the prevention of physical and social disabilities.
- BCG vaccination only to prevent leprosy: Not recommended.

3.5.9b Control measures

1) Report to local health authority.
2) Isolation: Not recommended. No restrictions in employment or attendance at school are indicated.
3) Quarantine: Not applicable.
4) Immunization of contacts: Not recommended
5) Investigation of contacts and source of infection: The initial examination of close contacts can be useful.

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3.5.10 Management of the disease\textsuperscript{61}

- In 1981, a World Health Organization (WHO) Study Group recommended multidrug therapy (MDT). MDT consists of 3 drugs: dapsone, rifampicin and clofazimine and this drug combination kills the pathogen and cures the patient.
- Since 1995, WHO provides free MDT for all patients in the world, initially through the drug fund provided by the Nippon Foundation and since 2000, through the MDT donation provided by Novartis and the Novartis Foundation for Sustainable Development.

\textsuperscript{61} WHO. “Leprosy Fact Sheet.” February 2010. \url{http://www.who.int/mediacentre/factsheets/fs101/en/index.html}
3.6 Mumps

ICD-10 B26

3.6.1 Identification

An acute viral disease characterized by fever, swelling and tenderness of one or more salivary glands, usually the parotid and sometimes the sublingual or submaxillary glands. Not all cases of parotitis are caused by mumps infection, but other parotitis-causing agents do not produce parotitis on an epidemic scale. As many as 40%–50% of mumps infections have been associated with respiratory symptoms, particularly in children under 5 years. About one-third of exposed susceptible people have inapparent infections; most infections in children under 2 are subclinical.

Mumps can cause sensorineural hearing loss in both children and adults. Pancreatitis, usually mild, occurs in 4% of cases; a suggested association with diabetes remains unproven.

Orchitis, most commonly unilateral, occurs in 20%–30% of affected postpubertal males. Testicular atrophy occurs in about one-third of patients, but sterility is extremely rare. Mumps orchitis has been reported to be a risk factor for testicular cancer.

Symptomatic aseptic meningitis occurs in up to 10% of mumps cases; patients usually recover without complications, though many require hospitalization. Mumps encephalitis is rare (1–2/10 000 cases), but can result in permanent sequelae, such as paralysis, seizures and hydrocephalus; the case-fatality rate for mumps encephalitis is about 1%.

Mumps infection during the first trimester of pregnancy is associated with a high (25%) incidence of spontaneous abortion, but there is no firm evidence that mumps during pregnancy causes congenital malformations.

Lab diagnosis:
Acute mumps infection can be confirmed through: a positive serological test for mumps-specific IgM antibodies, by seroconversion or by a significant (at least 4-fold) rise in serum mumps IgG titer as determined by standard serological assay; or through isolation of mumps virus from an appropriate clinical specimen (throat swab, urine, CSF). In research settings, typing methods can distinguish wild-type mumps virus from vaccine virus.

3.6.2 Infectious agent

Mumps virus, a member of the family *Paramyxoviridae*, genus *Rubulavirus*.

3.6.3 Occurrence

In temperate climates, winter and spring are peak seasons.
3.6.4 Reservoir

Humans.

3.6.5 Mode of transmission

Airborne or droplet spread; also direct contact with the saliva of an infection person.

3.6.6 Incubation period

16-18 days (Range: 14-25 days).

3.6.7 Period of communicability

Virus has been isolated from saliva (7 days before to 9 days after the onset of parotitis) and from urine (6 days before to 15 days after the onset of parotitis). Maximum infectiousness occurs between 2 days before to 4 days after onset of illness. Inapparent infections can be communicable.

3.6.8 Susceptibility and resistance

Immunity is generally lifelong and develops after either inapparent or clinical infections.

In the absence of immunization mumps is endemic, with an annual incidence usually greater than 100 per 100 000 population and epidemic peaks every 2–5 years. Serosurveys conducted prior to mumps vaccine introduction found that in some countries 90% of persons were immune by age 15 years, while in other countries a large proportion of the adult population remained susceptible. In countries were mumps vaccine has not been introduced, the incidence of mumps remains high, mostly affecting children 5–9 years. By the end of 2002, 121 countries/territories included mumps vaccine in their national immunization schedule. In countries where mumps vaccine coverage has been sustained at high levels the incidence of the disease has dropped tremendously.

3.6.9 Methods of control

3.6.9a Preventive measures

Immunization: Public education should encourage mumps immunization for susceptible individuals. Mumps vaccination is recommended at age 12–18 months, as part of MMR.

3.6.9b Control measures

- Report to local health authority.

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Mumps in Iraq

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Communicable Disease Control Guidelines

- Isolation: Respiratory isolation for 9 days from onset of parotitis. Exclusion from school or workplace until 9 days after onset of parotitis if susceptible contacts (those not immunized) are present.
- Concurrent disinfection: Of articles soiled with nose and throat secretions.
- Quarantine: Exclusion of susceptible from school or the workplace from the 12th through the 25th day after exposure if other susceptible are present.
- Immunization of contacts: Immunization after exposure may not always prevent infection. IG is not effective and not recommended.
- Investigation of contacts and source of infection: Immunization of susceptible contacts.

3.6.9c Epidemic measures
Immunize susceptible, especially those at risk of exposure. Serological screening to identify susceptible is impractical and unnecessary, since there is no risk in immunizing those who are already immune.

3.6.10 Management of the disease
- There is no specific treatment for mumps. The virus usually causes mild disease in children, but in adults can lead to complications, such as meningitis and orchitis. Mumps can be prevented by immunization.

Child with Mumps

63 Centers for Disease Control and Prevention's Public Health Image Library
3.7 Schistosomiasis

ICD-10 B65

3.7.1 Identification

In endemic areas, visible hematuria or positive reagent strip for hematuria, or with eggs of schistosomiasis hematobium in urine.

Case classification

- **Probable case**: Any visible hematuria associated with abdominal pain.
- **Suspected case**: Any terminal hematuria with the reagent strip associated with abdominal pain.
- **Confirmed case**: Microscopic examination showing eggs of *schistosomiasis hematobium* in urine.

3.7.2 Infectious agent

*Schistosoma hematobium*

3.7.3 Occurrence

*S. hematobium* is found in Africa and the Middle East. In Iraq it is found in the middle and southern regions. The risks groups are farmers and children ages 5-19 years old.

3.7.4 Reservoir

Humans are the principal reservoir.

3.7.5 Mode of transmission

Infection is acquired from water containing free swimming larval forms (cercariae) that have developed in snails. The eggs leave the body mainly in the urine and hatch in water and the liberated larvae (miracidia) penetrate into suitable freshwater snail hosts. After several weeks, the cercariae emerge from the snail and penetrate human skin, usually while the person is working, swimming or wading in water; they enter the bloodstream, are carried to blood vessels of the lungs, migrate to the liver, develop to maturity and then migrate to veins of the abdominal cavity. Adult forms migrate through anastomoses into the vesical plexus of the urinary bladder and eggs are deposited in venules and escape into the lumen of urinary bladder.
3.7.6 Incubation period

Acute systemic manifestations (Katayama fever) may occur in primary infections 2-6 weeks after exposure, immediately preceding and during initial egg deposition.

3.7.7 Period of communicability

Not communicable from person to person, but people with chronic Schistosomiasis may spread the infection by discharging eggs in urine into bodies of water for as long as they excrete eggs; it is common for the infection to last in excess of 10 years. Infected snails will release cercariae for as long as they live, a period that may last from several weeks to about 3 months.

3.7.8 Susceptibility and resistance

Susceptibility is universal any resistance developing as a result of infection is variable and poorly defined.

3.7.9 Methods of control

3.7.9a Preventive measures

a) Education of the population in endemic areas regarding the mode of transmission and methods of protection.
b) Safe disposal of urine so that viable eggs will not reach bodies of fresh water containing intermediate snail hosts.
c) Improvement of irrigation and agricultural practices; reduction of snail habitats by removing vegetation or by draining and filling.
d) Treatment of snail breeding sites with mollusicides.
e) Prevention of exposure to contaminated water (e.g., use of rubber boots). To minimize cercaria penetration after brief or accidental water exposure, towel dry skin surfaces that are wet with suspected water and apply 70% alcohol immediately to the skin to kill surface cercariae.
f) Provision of water for drinking, bathing and washing clothes from sources free of cercariae or treated to kill them. Effective measures for inactivating cercariae include water treatment with iodine or chlorine, or the use of paper filters. Allowing water to stand 48-72 hours before use is also effective.

3.7.9b Control measures

a) Treatment of patients in endemic areas with praziquantel to prevent disease progression and to reduce transmission by reducing egg passage.
b) Travelers visiting endemic areas should be advised of the risks and informed about preventive measures.
c) Reporting to local health authorities and centrally to CDC center by statistical forms.
3.7.9c Epidemic measures

a) Investigation of contacts and source of infection: Examination for Schistosomiasis and treatment of all who are infected and giving particular attention to children.

b) Provision of clean water, warning people against contact with water potentially containing cercariae and prohibiting contamination of water.

c) Treatment of areas that have high snail densities with mollusicides.

3.7.10 Management of the disease

- Praziquantel is the only available treatment against all forms of schistosomiasis. It is effective, safe and low-cost. Even though re-infection may occur after treatment, the risk of developing severe disease is diminished and even reversed when treatment is initiated in childhood.

- The WHO strategy for schistosomiasis control focuses on reducing disease through periodic, targeted treatment with praziquantel. This involves regular treatment of all people in at-risk groups. Treatment should be complemented with health education, as well as access to safe water and good sanitation.

- Groups targeted for treatment are:
  - school-aged children in endemic areas;
  - adults considered to be at risk in endemic areas, people with occupations involving contact with infested water – such as fishermen, farmers, irrigation workers – and women whose domestic tasks bring them into contact with infested water;
  - entire communities living in highly endemic areas.

- The frequency of treatment is determined by the prevalence of infection or visible hematuria (in the case of urogenital schistosomiasis) in school-age children. In high transmission areas, treatment may have to be repeated every year for several years.

- The aim is to reduce disease: periodic treatment of at-risk populations will cure mild symptoms and prevent infected people from developing severe, late-stage chronic disease.

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Skin vesicles on the forearm, created by the penetration

Source: CDC
3.8 Ascariasis

ICD-10 B77

3.8.1 Identification

Roundworm infection

**Case classification**

- **Suspected case**: A helminthic infection of the small intestine generally associated with few or no overt clinical symptoms.
- **Probable case**: Some patients have pulmonary manifestations caused by larval migration (mainly during reinfections) and characterized by wheezing, cough, fever, eosinophilia and pulmonary infiltration. Heavy parasite burdens may aggravate nutritional deficiency and, if chronic, may affect work and school performance. Serious complications, sometimes fatal, include bowel obstruction by a bolus of worms, particularly in children; or obstruction of bile duct, pancreatic duct or appendix by one or more adult worms. Reports of pancreatitis are increasing.
- **Confirmed case**: Diagnosis is made by identifying eggs in feces, or adult worms passed from the anus, mouth or nose. Intestinal worms may be visualized by radiological and sonographic techniques; pulmonary involvement may be confirmed by identifying *Ascaris* larvae in sputum or gastric washings.

3.8.2 Infectious agent

*Ascaris lumbricoides*

3.8.3 Occurrence

Common and worldwide, with greatest frequency in moist tropical countries where prevalence often exceeds 50%. Prevalence and intensity of infection are usually highest in children between 3 and 8 years.

3.8.4 Reservoir

Humans; ascarid eggs in soil.

3.8.5 Mode of transmission

Ingestion of infective eggs from soil contaminated with human feces or from uncooked produce contaminated with soil containing infective eggs, but not directly from person to person or from fresh feces. Transmission occurs mainly in the vicinity of the home, where children, in the absence of sanitary facilities, fecally pollute the area; heavy infections in children are frequently the result of ingesting soil (pica). Contaminated soil may be carried long distances on feet or footwear into houses and conveyances; transmission of infection by dust is also possible.
Eggs reach the soil from the feces, then undergo development (embryonation); at summer temperatures they become infective after 2-3 weeks and may remain infective for several months or years in favorable soil. Ingested embryonated eggs hatch in the intestinal lumen; the larvae penetrate the gut wall and reach the lungs via the circulatory system. Larvae grow and develop in the lungs, pass into the alveoli 9-10 days after infection, ascend the trachea and are swallowed to reach the small intestine 14-20 days after infection, where they grow to maturity, mate and begin laying eggs 45-60 days after initial ingestion of the embryonated eggs. Eggs passed by gravid females are discharged in feces.

3.8.6 Incubation period

Life cycle requires 4-8 weeks to complete.

3.8.7 Period of communicability

As long as mature fertilized female worms live in the intestine. Usual life span of adult worms is 12 months; maximum may reach 24 months. The female worm can produce more than 200,000 eggs a day. Under favorable conditions, embryonated eggs can remain viable in soil for years.

3.8.8 Susceptibility and resistance

Susceptibility is general.

3.8.9 Methods of control

3.8.9a Preventive measures

1) Educate the public in the use of toilet facilities.
2) Provide adequate facilities for proper disposal of feces and prevent soil contamination in areas immediately adjacent to houses, particularly in children's play areas.
3) In rural areas, construct latrines that prevent dissemination of ascarid eggs through overflow, drainage or otherwise. Treating human feces by composting for later use as fertilizer may not kill all eggs.
4) Encourage satisfactory hygienic habits on the part of children; in particular, train them to wash hands before eating and handling food.
5) In endemic areas, protect food from dirt. Food that has been dropped on the floor should not be eaten unless washed or reheated.
6) WHO recommends a strategy focused on high-risk groups for the control of morbidity due to soil-transmitted helminthes.

3.8.9b Control measures

1) Report to local health authority: Official report not ordinarily justifiable.
2) Isolation: Not applicable.
3) Concurrent disinfection: Sanitary disposal of feces.
4) Quarantine: Not applicable.
5) Immunization of contacts: Not applicable.
6) Investigation of contacts and source of infection.
7) Specific treatment: Single-dose oral mebendazole (500 mg), albendazole (400 mg, half dose for children 12-24 months); on theoretical grounds, both are contraindicated during the first trimester of pregnancy unless there are specific medical or public health indications. Erratic migration of ascariid worms has been reported following mebendazole therapy; this may also occur with other medications, or spontaneously in heavy infections. Single-dose pyrantel pamoate (10 mg/kg) or levamisole (2.5 mg/kg) also effective.

3.8.9c Epidemic measures
Survey for prevalence in highly endemic areas, educate the community in environmental sanitation and in personal hygiene and provide treatment facilities. Community treatment for high-risk groups, especially children or for the whole population.

3.8.10 Management of the disease\(^{66}\)
- Infected individuals (and domestic animals) should be treated with medicine to reduce disease transmission. Ascariasis can be effectively treated with mebendazole or pyrantel pamoate.

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http://www.who.int/water_sanitation_health/diseases/ascariasis/en/

\(^{67}\) http://www.dpd.cdc.gov/dpdx/images/ParasiteImages/A-F/Ascariasis/Ascaris_female.jpg
3.9 Ancylostomiasis (Hookworm disease)

ICD-10 B76

3.9.1 Identification

Hookworm disease (Ancylostomiasis, Uncinariasis, Necatoriasis)

Case classification

- **Suspect case:** A common chronic parasitic infection with a variety of symptoms, usually in proportion to the degree of anemia.
- **Probable case:** heavy infections, leads to iron deficiency and hypochromic, microcytic anemia, the major cause of disability. Children with heavy long-term infection may have hypoproteinemia and may be retarded in mental and physical development. Occasionally, severe acute pulmonary and GI reactions follow exposure to infective larvae.
- **Confirmed case:** finding hookworm eggs in feces; early stool examinations may be negative until worms mature. Species differentiation requires microscopic examination of larvae cultured from the feces, or examination of adult worms expelled by purgation following a vermifuge. PCR-RFLP techniques allow species differentiation.

3.9.2 Infectious agent

*Ancylostoma duodenale, A. ceylanicum, A. braziliense, A. caninum* and *Necator americanus*.

3.9.3 Occurrence

Endemic in tropical and subtropical countries where sanitary disposal of human feces is not practiced and soil, moisture and temperature conditions favor development of infective larvae. Also occurs in temperate climates under similar environmental conditions (e.g. mines). Both *Necator* and *Ancylostoma* occur in many parts of Asia (particularly southeastern Asia), the South Pacific and eastern Africa. *N. americanus* is the prevailing species throughout southeastern Asia, most of tropical Africa and America; *A. duodenale* prevails in North Africa, including the Nile Valley, northern India, northern parts of eastern Asia and the Andean areas of South America. *A. ceylanicum* occurs in southeastern Asia but is less common than either *N. americanus* or *A. duodenale*. *A. caninum* has been described in Australia as a cause of eosinophilic enteritis syndrome.

3.9.4 Reservoir

Humans for *A. duodenale* and *N. americanus*; cats and dogs for *A. ceylanicum, A. braziliense* and *A. caninum*. 
3.9.5 Mode of transmission

Eggs in feces are deposited on the ground and hatch; under favorable conditions of moisture, temperature and soil type, larvae develop to the third stage, becoming infective in 7-10 days. Human infection occurs when infective larvae penetrate the skin, usually of the foot; in so doing, they produce a characteristic dermatitis (ground itch). The larvae of *A. caninum* and *A. braziliense* die within the skin, having produced cutaneous larva migrans. Normally, the larvae of *Necator, A. duodenale, A. ceylanicum* and other *Ancylostoma* enter the skin and pass via lymphatics and bloodstream to the lungs, enter the alveoli, migrate up the trachea to the pharynx, are swallowed and reach the small intestine where they attach to the intestinal wall, developing to maturity in 6-7 weeks (3-4 weeks in the case of *A. ceylanicum*) and typically producing thousands of eggs per day. Infection with *Ancylostoma* may also be acquired by ingesting infective larvae; possible vertical transmission through breast milk has been reported.

3.9.6 Incubation period

Symptoms may develop after a few weeks to many months, depending on intensity of infection and iron intake of the host. Pulmonary infiltration, cough and tracheitis may occur during the lung migration phase of infection, particularly in *Necator* infections. After entering the body, *A. duodenale* may become dormant for about 8 months, after which development resumes, with a patent infection (stools containing eggs) a month later.

3.9.7 Period of communicability

No person-to-person transmission, but infected people can contaminate soil for several years in the absence of treatment. Under favorable conditions, larvae remain infective in soil for several weeks.

3.9.8 Susceptibility

Universal; no evidence that immunity develops with infection.

3.9.9 Methods of control

3.9.9a Preventive methods

a) Educate the public to the dangers of soil contamination by human, cat or dog feces, and in preventive measures, including wearing shoes in endemic areas.
b) Prevent soil contamination by installation of sanitary disposal systems for human feces, especially sanitary latrines in rural areas. Night soil and sewage effluents are hazardous, especially where used as fertilizer.
c) Examine and treat people migrating from endemic to receptive non endemic areas.
d) WHO recommends a strategy focused on high-risk groups for the control of morbidity due to soil-transmitted helminthes.
3.9.9b Control methods

a) Report to local health authority.
b) Isolation: Not applicable.
c) Concurrent disinfection: Safe disposal of feces to prevent contamination of soil.
d) Quarantine: Not applicable.
e) Immunization of contacts: Not applicable.
f) Investigate contacts and source of infection.
g) Specific treatment: Single dose oral treatment with mebendazole, albendazole (half dose for children 12-24 months), levamisole or pyrantel pamoate is recommended; adverse reactions are infrequent. Follow-up stool examination is indicated after 2 weeks, and treatment must be repeated if a heavy worm burden persists. Iron supplementation will correct the anemia and should be used in conjunction with deworming. Transfusion may be necessary for severe anemia. As a general rule, pregnant women should not be treated in the first trimester unless there are specific medical or public health reasons.

3.9.9c Epidemic measures

Prevalence survey in highly endemic areas: provide periodic mass treatment. Health education in environmental sanitation and personal hygiene, and provide facilities for excreta disposal.

3.9.10 Management of the disease

- Efforts to control hookworm infection include the sanitary disposal of feces and educational campaigns about the proper use of latrines. At this time, the most cost-effective way to control hookworm infection has been through population-wide treatment with either albendazole or mebendazole.

69 CDC’s Public Health Image Library
3.10 Trichuriasis

ICD-10 B79

3.10.1 Identification

Whipworm disease

Case classification:
- **Suspected case:** A nematode infection of the large intestine, usually asymptomatic.
- **Probable case:** Heavy infections may cause bloody, mucoid stools and diarrhea. Rectal prolapse, clubbing of fingers, hypoproteinemia, anemia and growth retardation may occur in heavily infected children.
- **Confirmed case:** demonstration of eggs in feces or sigmoidoscopic observation of worms attached to the wall of the lower colon in heavy infections.

3.10.2 Infectious agent

*Trichuris trichiura*

3.10.3 Occurrence

Worldwide, especially in warm, moist regions.

3.10.4 Reservoir

Humans

3.10.5 Mode of transmission

Indirect, particularly through pica or ingestion of contaminated vegetables; no immediate person-to-person transmission. Eggs passed in feces require a minimum of 10-14 days in warm moist soil to become infective. Hatching of larvae follows ingestion of infective eggs from contaminated soil, attachment to the mucosa of the caecum and proximal colon, and development into mature worms. Eggs appear in the feces 70-90 days after ingestion of embryonated eggs; symptoms may appear much earlier.

3.10.6 Incubation period

Indefinite
3.10.7 Period of communicability

Several years in untreated carriers.

3.10.8 Susceptibility and resistance

Universal.

3.10.9 Methods of control

3.10.9a Preventive measures

1) Educate all members of the family, particularly children, in the use of toilet facilities.
2) Provide adequate facilities for feces disposal.
3) Encourage satisfactory hygienic habits, especially hand washing before food handling; avoid ingestion of soil by thorough washing of vegetables and other foods contaminated with soil.
4) WHO recommends a strategy focused on high-risk groups for the control of morbidity due to soil-transmitted helminthes, including community treatment.

3.10.9b Control measures

1) Report to local health authority.
2) Isolation: Not applicable.
3) Concurrent disinfection: Sanitary disposal of feces.
4) Quarantine: Not applicable.
5) Immunization of contacts: Not applicable.
6) Investigation of contacts and source of infection: Examine feces of all symptomatic members of the family group, especially children and playmates.
7) Specific treatment: Mebendazole is the drug of choice. Albendazole (half dose for children 12-24 months), and pyrantel are alternative drugs.

3.10.10 Management of the disease\(^{70}\)

- Mebendazole taken by mouth for 3 days is commonly prescribed when the infection causes symptoms. Albendazole (half dose for children 12-24 months) and pyrantel are used as alternative therapies.

Trichuris trichiura egg
3.11 Strongyloidiosis

ICD-10 B78

3.11.1 Identification

Case classification:

- **Suspected Case:** asymptomatic helminthic infection of the duodenum and upper jejunum. Clinical manifestations include transient dermatitis when larvae of the parasite penetrate the skin on initial infection;
- **Probable Case:** cough, and sometimes demonstrable pneumonitis, abdominal pain (usually epigastric, diarrhea and urticaria; nausea, weight loss, vomiting, weakness and constipation.
- **Confirmed Case:** identifying larvae in concentrated stool specimens (motile in freshly passed feces), in duodenal aspirates or, occasionally, in sputum. Feces may show developing stages of the parasite, Serological tests based on larval stage antigens are positive in 80%-85% of infected patients.

3.11.2 Infectious agent

*Strongyloides stercoralis*

3.11.3 Occurrence

Throughout tropical and temperate areas; more common in warm, wet regions.

3.11.4 Reservoir

Humans are the principal reservoir, with occasional transmission of dog and cat strains to humans.

3.11.5 Mode of transmission

Infective (filariform) larvae develop in feces or moist soil contaminated with feces, penetrates the skin, enters the venous circulation and are carried to the lungs. They penetrate capillary walls, enter the alveoli, ascend the trachea to the epiglottis and descend into the digestive tract to reach the upper part of the small intestine, where development of the adult female is completed. The adult worm, female, lives embedded in the mucosal epithelium of the intestine, especially the duodenum, where eggs are deposited. These hatch and liberate rhabditiform (non infective) larvae that migrate into the intestinal lumen, exit in feces and develop after reaching the soil into either infective filariform larvae (which may infect the same or a new host) or free-living male and female adults. The free-living fertilized females produce eggs that hatch and liberate rhabditiform larvae, which may become filariform larvae within 24-36 hours. In some
individuals, rhabditiform larvae may develop to the infective stage before leaving the body and penetrate through the intestinal mucosa or perianal skin; the resulting autoinfection can cause persistent infection for many years.

3.11.6 Incubation period

From penetration of the skin by filariform larvae until rhabditiform larvae appear in the feces takes 2-4 weeks; the period until symptoms appear is indefinite and variable.

3.11.7 Period of communicability

As long as living worms remain in the intestine; up to 35 years in cases of autoinfection.

3.11.8 Susceptibility and resistance

Universal.

3.11.9 Methods of control

3.11.9a Preventive measures

1) Dispose of human feces in a safe manner.
2) Attention to hygienic habits.
3) Examine and treat infected dogs, cats and monkeys in contact with people.

3.119b Control measures

1) Report to local health authority.
2) Isolation: Not applicable.
3) Concurrent disinfection: Safe disposal of feces.
4) Quarantine: Not applicable.
5) Immunization of contacts: Not applicable.
6) Investigation of contacts and source of infection.
7) Specific treatment: Because of the potential for autoinfection and dissemination, all infections, regardless of worm burden, should be treated.

3.11.10 Management of the disease

Ivermectin, thiabendazole and albendazole are the most effective medicines for treating the infection. Albendazole is considered the least effective. Ivermectin, the drug of choice, is not available in all endemic countries. Moreover, the optimal schedule has yet to be defined.

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71 WHO. “Neglected tropical diseases: Strongyloidosis.”
http://www.who.int/neglected_diseases/diseases/strongyloidiasis/en/
Larva of *Strongyloides stercoralis*, the roundworm that causes strongyloidiasis
3.12 Enterobiasis

ICD-10 B80

3.12.1 Identification

Pinworm disease

**Case classification:**

- **Suspected Case:** A common intestinal helminthic infection that is often asymptomatic. There may be perianal itching, disturbed sleep, irritability and sometimes secondary infection of the scratched skin.
- **Probable Case:** Other clinical manifestations include vulvovaginitis, salpingitis, and pelvic and liver granulomata. Appendicitis and enuresis have rarely been reported as possible associated conditions.
- **Confirmed Case:** applying transparent adhesive tape (tape swab or pinworm paddle) to the perianal region and examining the tape or paddle microscopically for eggs; the material is best obtained in the morning before bathing or passage of stools. Examination should be repeated 3 or more times before accepting a negative result. Eggs are sometimes found on microscopic stool and urine examination. Female worms may be found in feces and in the perianal region during rectal or vaginal examinations.

3.12.2 Infectious agent

*Enterobius vermicularis*, an intestinal nematode.

3.12.3 Occurrence

Worldwide, affecting all socioeconomic classes, with high rates in some areas. It is the most common worm infection in other countries of temperate climate; prevalence is highest in school-age children (in some groups near 50%). Infection often occurs in more than one family member.

3.12.4 Reservoir

Humans. Pinworms of other animals are not transmissible to humans.

3.12.5 Mode of transmission

Direct transfer of infective eggs by hand from anus to mouth of the same or another person, or indirectly through clothing, bedding, food or other articles contaminated with parasite eggs. Dust borne infection is possible in heavily contaminated households and institutions. Eggs become
infective within a few hours after being deposited at the anus by migrating gravid females.

3.12.6 Incubation period

The life cycle requires 2-6 weeks. Symptomatic disease with high worm burdens results from successive reinfection occurring within months following initial exposure.

3.12.7 Period of communicability

As long as gravid females discharge eggs on perianal skin. Eggs remain infective in an indoor environment for about 2 weeks.

3.12.8 Susceptibility and resistance

Universal. Differences in frequency and intensity of infection are due primarily to differences in exposure.

3.12.9 Methods of control

3.12.9a Preventive measures

1) Educate the public in personal hygiene, particularly the need to wash hands before eating or preparing food. Keep nails short; discourage nail biting and scratching anal area.
2) Remove sources of infection through treatment of cases.
3) Daily morning bathing, with showers (or stand-up baths) preferred to tub baths.
4) Change to clean underclothing, nightclothes and bed sheets frequently, preferably after bathing.
5) Clean and vacuum house daily for several days after treatment of cases.
6) Reduce overcrowding in living accommodations.
7) Provide adequate toilets; maintain cleanliness in these facilities.

3.12.9b Control measures

1) Report to local health authority: Official report not ordinarily justifiable.
2) Isolation: Not applicable.
3) Concurrent disinfection: Change bed linen and underwear of infected person daily for several days after treatment, avoiding aerial dispersal of eggs. Use closed sleeping garments. Eggs on discarded linen are killed by exposure to temperatures of 55°C (131°F) for a few seconds; either boil bed clothing or use a washing machine on the "hot" cycle. Clean and vacuum sleeping and living areas daily for several days after treatment.
4) Quarantine: Not applicable.
5) Immunization of contacts: Not applicable
6) Investigation of contacts and source of infection: Examine all members of an affected family or institution.
7) Specific treatment: Pyrantel pamoate, mebendazole or albendazole. Treatment to be repeated after 2 weeks; concurrent treatment of the whole family may be advisable if several members are infected.

3.12.9c Epidemic measures
Multiple cases in schools and institutions can best be controlled through systematic treatment of all infected individuals and household contacts.

3.12.10 Management of the disease72

- The main treatment is a single dose of either mebendazole or albendazole, which kill the pinworms (not the eggs).
- More than one household member is likely to be infected, so the entire household is often treated. The single-dose treatment is often repeated after 2 weeks. This treats worms that hatched since the first treatment.
- To control the eggs:
  - Clean toilet seats daily
  - Keep fingernails short and clean
  - Wash all bed linens twice a week
  - Wash hands before meals and after using the toilet
- Avoid scratching the infected area around the anus. This can cause contamination and fuel transmission.
- Keep hands and fingers away from nose and mouth unless freshly washed. Carry out these measures while family members are being treated for pinworms.

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3.13 Cestodiasis (Hymenolepis)

ICD-10 B71.0

3.13.1 Identification

Dwarf tapeworm infection.

Case classification:
- **Suspected Case**: abdominal pain and other vague symptoms such as pallor, loss of weight and weakness.
- **Probable Case**: An intestinal infection with very small tapeworms; light infections are usually asymptomatic. Massive numbers of worms may cause enteritis with or without diarrhea.
- **Confirmed Case**: Microscope identification of eggs in feces confirms diagnosis.

3.13.2 Infectious agent

*Hymenolepis nana* (dwarf tapeworm), the only human tapeworm without an obligatory intermediate host.

3.13.3 Occurrence

Cosmopolitan; more common in warm than cold, and in dry than wet climates. Dwarf tapeworm is the most common human tapeworm in the USA and Latin America; it is common in Australia, Mediterranean countries, the Near East and India.

3.13.4 Reservoir

Humans, possibly mice.

3.13.5 Mode of transmission

Eggs of *H. nana* are infective when passed in feces. Infection is acquired through ingestion of eggs in contaminated food or water; directly from fecally contaminated fingers (autoinfection or person-to-person transmission); or ingestion of insects bearing larvae that have developed from eggs ingested by the insect. *H. nana* eggs, once ingested, hatch in the intestine, liberating oncospheres that enter mucosal villi and develop into cysticercoids; these rupture into the lumen and grow into adult tapeworms. Some H nana eggs are immediately infectious when released from the proglottids in the human gut, so autoinfection’s or person-to-person transmission can occur.
3.13.6 Incubation period

Onset of symptoms is variable; the development of mature worms requires about 2 weeks.

3.13.7 Period of communicability

As long as eggs are passed in feces. Infection may persist for years.

3.13.8 Susceptibility and resistance

Universal; infection produces resistance to reinfection. Children are more susceptible than adults; intensive infection occurs in immunodeficient and malnourished children.

3.13.9 Methods of control

3.13.9a Preventive measures

1) Educate the public in personal hygiene and safe disposal of feces.
2) Provide and maintain clean toilet facilities.
3) Protect food and water from contamination with human and rodent feces.
4) Treat to remove sources of infection.
5) Eliminate rodents from home environment.

3.13.9b Control measures

1) Report to local health authority: Official report not ordinarily justifiable, Class 5 (see Reporting).
2) Isolation: Not applicable.
3) Concurrent disinfection: Safe disposal of feces.
4) Quarantine: Not applicable.
5) Immunization of contacts: Not applicable.
6) Investigation of contacts and source of infection: Fecal examination of family or institution members.

3.13.9c Epidemic measures

Outbreaks in schools and institutions can best be controlled through treatment of infected individuals and special attention to personal and group hygiene.

3.13.10 Management of the disease

Specific treatment: Praziquantel or niclosamide is effective. Albendazole may be considered where intestinal helminthiases coexist.
Beef tapeworm
3.14 Taeniasis (taenia saginata)

ICD-10 B68.0: Taenia Solium Taeniasis
ICD-10 B68.1: Taenia Saginata Taeniasis
ICD-10 B69: Cysticercosis

3.14.1 Identification

Beef tapeworm and pork tapeworm.

Case classification:

- **Suspect case**: Clinical manifestations of infection with the adult worm, if present, are variable and may include nervousness, insomnia, anorexia, weight loss, abdominal pain and digestive disturbances.
- **Probable case**: In the presence of somatic cysticercosis, epileptiform seizures, headache, signs of intracranial hypertension or psychiatric disturbances strongly suggest cerebral involvement Neurocysticercosis.
- **Confirmed case**: Infection with an adult tapeworm is diagnosed by identification of proglottids (segments), eggs or antigens of the worm in the feces or on anal swabs. Specific diagnosis is based on the morphology of the scolex (head) and/or gravid proglottids.

3.14.2 Infectious agent

*Taenia solium*, the pork tapeworm, causes both intestinal infection with the adult worm and extra intestinal infection with the larvae (cysticerci). *T. saginata*, the beef tapeworm, only causes intestinal infection with the adult worm in humans.

3.14.3 Occurrence

Worldwide; frequent wherever beef or pork is eaten raw or insufficiently cooked and where sanitary conditions are lacking.

3.14.4 Reservoir

Humans are the definitive host of both species. Cattle are the intermediate hosts for *T. saginata* and pigs for *T. solium*.

3.14.5 Mode of transmission

Eggs of *T. saginata* passed in the stool of an infected person are infectious only to cattle, in the flesh of which the parasites develop into cysticercus bovis, the larval stage of *T. saginata*. In
humans, infection follows ingestion of raw or undercooked beef containing cysticerci; adult worm develops in the intestine.

3.14.6 Incubation period

Symptoms of cysticercosis may appear from weeks to 10 years or more after infection. Eggs appear in the stool 8-12 weeks after infection with the adult *T. solium* tapeworm, 10-14 weeks with *T. saginata*.

3.14.7 Period of communicability

*T. saginata* is not directly transmitted from person to person, but *T. solium* may be. Eggs of both species are disseminated into the environment as long as the worm remains in the intestine, sometimes more than 30 years; eggs may remain viable in the environment for months.

3.14.8 Susceptibility and resistance

Susceptibility is general. No apparent resistance follows infection; the presence of more than one tapeworm in a person has rarely been reported.

3.14.9 Methods of control

3.14.9a Preventive measures

1) Educate the public to prevent fecal contamination of soil, water, and human and animal food; to avoid use of sewage effluents for pasture irrigation; and to cook beef and pork thoroughly.
2) Appropriate measures to protect patients from themselves and their contacts are necessary.
3) Freezing beef at a temperature below -5°C (23°F) for more than 4 days kills the cysticerci effectively.
4) Inspection of the carcasses of cattle and swine will detect only a proportion of infected carcasses; these should be condemned, irradiated or processed into cooked products.
5) Prevent swine access to latrines and human feces.

3.14.9b Control measures

1) Report to local health authority: Selectively reportable.
2) Isolation: Not applicable. Stools of patients with untreated taeniasis due to *T. solium* may be infective.
3) Concurrent disinfection: Dispose of feces in a sanitary manner; emphasize strict sanitation, with hand washing after defecating and before eating, especially for *T. solium*.
4) Quarantine: Not applicable.
5) Immunization of contacts: Not applicable.
6) Investigation of contacts and source of infection: Evaluate symptomatic contacts.
7) Specific treatment: Praziquantel is effective in the treatment of *T. saginata* and *T. solium* intestinal infections. Niclosamide, no longer widely available, is an alternative.

### 3.14.10 Management of the disease

- Tapeworms are treated with medications taken by mouth, usually in a single dose. The drug of choice for tapeworm infections is praziquantel. Niclosamide can also be used. Patients with active CNS cysticercosis may benefit from treatment with praziquantel or albendazole under hospitalization; a short course of corticosteroids is usually given to control cerebral edema due to dying cysticerci. Where cysticidal treatment is not indicated, symptomatic treatment, such as with antiepileptic drugs, may bring relief. In some cases surgical intervention may be needed to relieve symptoms.
3.15 Amebiasis

ICD-10 A06

3.15.1 Identification

Case classification:

- **Suspected Case:** A protozoan parasite infection that exists in 2 forms: the hardy infective cyst and the more fragile potentially pathogenic trophozoite. The parasite may act as a commensal or invade the tissues and give rise to intestinal or extra intestinal disease.
- **Probable Case:** Most infections are asymptomatic. Intestinal disease varies from acute or fulminating dysentery with fever, chills and bloody or mucoid diarrhea (amoebic dysentery), to mild abdominal discomfort with diarrhea containing blood or mucus.
- **Confirmed case:** microscopic demonstration of trophozoites or cysts in fresh or suitably preserved fecal specimens.

3.15.2 Infectious agent

*Entamoeba histolytica.*

3.15.3 Occurrence

Amebiasis is mostly a disease of young adults; liver abscesses occur predominantly in males. Amebiasis is rare below age 5 and especially below age 2, when dysentery is due typically to shigellae. Prevalence rates of cyst passage, usually based on cyst morphology, vary from place to place, with rates generally higher in areas with poor sanitation. In areas with good sanitation, amoebic infections tend to cluster in households and institutions.

3.15.4 Reservoir

Humans

3.15.5 Mode of transmission

Mainly through ingestion of fecally contaminated food or water containing amoebic cysts, which are relatively chlorine resistant. Patients with acute amoebic dysentery probably pose only limited danger to others because of the absence of cysts in dysenteric stools and the fragility of trophozoites.

3.15.6 Incubation period
From a few days to several months or years; commonly 2-4 weeks.

3.15.7 Period of communicability

During the period *E. histolytica* are passed, which may continue for years.

3.15.8 Susceptibility and resistance

General. Susceptibility to reinfection has been demonstrated but is apparently rare.

3.15.9 Methods of control

3.15.9a Preventive measures

1) Educate the general public in personal hygiene, particularly in sanitary disposal of feces and in hand washing after defecation and before preparing or eating food. Disseminate information regarding the risks involved in eating uncleaned or uncooked fruits and vegetables and in drinking water of questionable purity.
2) Dispose of human feces in a sanitary manner.
3) Protect public water supplies from fecal contamination.
4) Treat known carriers; stress the need for thorough hand-washing after defecation to avoid reinfection from an infected domestic resident.
5) Health agencies should supervise the sanitary practices of people who prepare and serve food in public eating places and the general cleanliness of the premises involved. Routine examination of food handlers as a control measure is impractical.
6) Disinfectant dips for fruits and vegetables are of unproven value in preventing transmission of *E. histolytica*. Thorough washing with potable water and keeping fruits and vegetables dry may help; cysts are killed by desiccation, by temperatures above 50°C (122°F) and by irradiation.
7) Use of chemo prophylactic agents is not advised.

3.15.9b Control measures

1) Report to local health authority: In selected endemic areas.
2) Isolation: For hospitalized patients, enteric precautions in the handling of feces, contaminated clothing and bed linen. Exclusion of individuals infected with *E. histolytica* from food handling and from direct care of hospitalized and institutionalized patients. Release to return to work in a sensitive occupation when chemotherapy is completed.
3) Concurrent disinfection: Sanitary disposal of feces.
4) Quarantine: Not applicable.
5) Immunization of contacts: Not applicable.
6) Investigation of contacts and source of infection: Household members and other suspected contacts should have adequate microscopic examination of feces.
3.15.9c Epidemic measures
Any group of possible cases requires prompt laboratory confirmation to exclude false-positive identification of *E. histolytica* or other causal agents and epidemiological investigation to determine source of infection and mode of transmission. If a common vehicle is indicated, such as water or food, appropriate measures should be taken to correct the situation.

3.15.9d Disaster implications
Disruption of normal sanitary facilities and food management will favor an outbreak of amebiasis, especially in populations that include large numbers of cyst passers.

3.15.10 Management of the disease
Specific treatment: Acute amoebic dysentery should be treated with metronidazole. In cases of extra intestinal Amoebiasis or refractory intestinal amebiasis, metronidazole should be followed by iodoquinol, paromomycin or diloxanide furoate. Tinidazole and ornidazole are also useful single-dose treatments against luminal and tissue disease. If a patient with a liver abscess continues to be febrile after 72 hours of metronidazole treatment, nonsurgical aspiration may be indicated. Chloroquine is sometimes added to metronidazole or dehydroemetine for treating a refractory liver abscess. Abscesses may require surgical aspiration if there is a risk of rupture or if the abscess continues to enlarge despite treatment. Asymptomatic carriers may be treated with iodoquinol, paromomycin or diloxanide furoate. Metronidazole is not recommended for use during the first trimester of pregnancy.
3.16 Giardiasis

ICD-10 A07.1

3.16.1 Identification

Case classification:

- **Suspected case:** A protozoan infection principally of the upper small intestine; it can be asymptomatic; or bring on acute, self-limited diarrhea; or lead to intestinal symptoms such as chronic diarrhea.
- **Probable case:** steatorrhea, abdominal cramps, bloating, frequent loose and pale greasy stools, fatigue, malabsorption (of fats and fat-soluble vitamins) and weight loss. There is usually no extra intestinal invasion, but reactive arthritis and, in severe giardiasis, damage to duodenal and jejunal mucosal cells may occur.
- **Confirmed case:** identification of cysts or trophozoites in feces (to rule out the diagnosis at least 3 negative results are needed). Because Giardia infection is usually asymptomatic, the presence of *G. lamblia* (in stool or duodenum) does not necessarily indicate that Giardia is the cause of illness. Tests using ELISA or direct fluorescent antibody methods to detect antigens in the stool, generally more sensitive than direct microscopy, are commercially available. Where results of stool examination and antigen assays are questionable, it may be useful to examine for trophozoites from duodenal fluid (aspiration or string test) or mucosa obtained by small intestine biopsy.

3.16.2 Infectious agent

*Giardia lamblia* (*G. intestinalis, G. duodenalis*), a flagellate protozoan.

3.16.3 Occurrence

Worldwide. Children are infected more frequently than adults. Prevalence is higher in areas of poor sanitation and institutions with children not toilet trained, including day care centers. The prevalence of stool positivity in different areas may range between 1% and 30%.

3.16.4 Reservoir

Humans; possibly beaver and other wild and domestic animals.

3.16.5 Mode of transmission

Person-to-person transmission occurs by hand-to-mouth transfer of cysts from the feces of an infected individual, especially in institutions and day care centers; this is probably the principal mode of spread. Anal intercourse also facilitates transmission.
3.16.6 Incubation period

Usually 3-25 days or longer; median 7-10 days.

3.16.7 Period of communicability

Entire period of infection, often months.

3.16.8 Susceptibility and resistance

Asymptomatic carrier rate is high; infection is frequently self-limited. Pathogenicity of *G. lamblia* for humans has been established by clinical studies. Persons with HIV infection may have more serious and prolonged giardiasis.

3.16.9 Methods of control

3.16.9a Preventive measures

1) Educate families, personnel and inmates of institutions, and especially adult personnel of day care centers, in personal hygiene and the need for washing hands before handling food, before eating and after toilet use.
2) Filter public water supplies exposed to human or animal fecal contamination.
3) Protect public water supplies against contamination with human and animal feces.
4) Dispose of feces in a sanitary manner.
5) Boil emergency water supplies. Chemical treatment with hypochlorite or iodine less reliable; use 0.1 to 0.2 ml (2 to 4 drops) of household bleach or 0.5 ml of 2% tincture of iodine per liter for 20 minutes (longer if water is cold or turbid).

3.16.9b Control measures

1) Report to local health authority: Case report in selected areas.
2) Isolation: Enteric precautions.
3) Concurrent disinfection of feces and articles soiled there-with. In communities with a modern and adequate sewage disposal system, feces can be discharged directly into sewers without preliminary disinfection. Terminal cleaning.
4) Quarantine: Not applicable.
5) Immunization of contacts: Not applicable.
6) Investigation of contacts and source of infection: Microscopic examination of feces of household members and other suspected contacts, especially if symptomatic.

3.16.9c Epidemic measures
Institute an epidemiological investigation of clustered cases in an area or institution to determine source of infection and mode of transmission. A common vehicle, such as water, food or association with a day care center or recreational area must be sought; institute applicable preventive or control measures. Control of person-to-person transmission requires special emphasis on personal cleanliness and sanitary disposal of feces.

3.16.10 Management of the disease

Specific treatment: 5-nitroimidazoles: one daily dose of 2 grams metronidazole (children 15 mg/kg) for 3 days, or tinidazole 2 grams in a single dose (children 50-75 mg/kg) are the drugs of choice. Furazolidone is available in pediatric suspension for young children and infants (2 mg/kg thrice daily for 7-10 days). Paromomycin can be used during pregnancy, but when disease is mild, delay of treatment till after delivery is recommended. Drug resistance and relapses may occur with any drug.
3.17 Acute Lower Respiratory Infections (ALRI)

ICD-10 A49

3.17.1 Identification

Pneumonia
Symptoms: Cough or difficult breathing;  
Signs: 50 or more breaths per minute for infants age 2 months up to 1 year, or 40 or more breaths per minute for children age 1 up to 5 years old; and no chest in drawing, general danger signs, strider in calm child or severe malnutrition.

Severe pneumonia
Symptoms: Cough or difficult breathing and  
Signs: Chest in drawing and strider in a calm child or severe malnutrition.

Very severe disease
Symptoms: Cough or difficult breathing  
Signs: General danger signs: unable to drink or breast feed; convulsions; abnormally sleepy or difficult to wake, strider in a calm child or severe malnutrition. 

Infants below 2 months of age: Cases are classified as either “Severe Pneumonia” or “Very severe disease”, as the illness may progress rapidly in this age group and it may be difficult to differentiate “pneumonia” from other severe conditions requiring inpatient hospital management.

3.17.2 Infectious agent

Bacteria: the most common are likely to be *Streptococcus pneumonia* and *Homophiles influenza* (and *Staphylococcus aureus* to a lesser extent).

3.17.3 Occurrence

Low temperatures, especially in the North, may increase the relative risk of children’s acquiring pneumonia.

3.17.4 Reservoir

Humans are only known reservoir.

3.17.5 Mode of transmission

Airborne: respiratory droplets.
3.17.6 Incubation period

Depends on the infective agent. Usually 2-5 days.

3.17.7 Period of communicability

Depends on the infective agent. Usually during the symptomatic phase.

3.17.8 Susceptibility and resistance

Pneumonia can occur in anyone. It occurs with increased frequency in individuals whose immune systems are deficient such as malnourished, HIV infected, with diabetes, underlying lung diseases, cancers, and treatment with immunosuppressive drugs. Infants and very young children are highly vulnerable, as are the elderly.

3.17.9 Methods of control

3.17.9a Preventive measures

1) Immunization against measles, diphtheria, and whooping cough are effective in reducing impact of ALRI.
2) Health education on early danger signs for prompt care-seeking.
3) Adequate feeding, including exclusive breastfeeding to avoid malnutrition.

3.17.9b Control measures

- Priority is early recognition and adequate treatment of cases
- First-line antibiotic for cases in under-fives classified as pneumonia is amoxycillin; second-line antibiotics are cotrimoxazole, ampicillin and, used less frequently, procaine penicillin. The IMCI guidelines under development propose the use of cephalexin and erythromycin as first and second line antibiotics, respectively, for young infants; and intramuscular cefotaxime as pre-referral antibiotic for severe under-five cases that cannot take oral antibiotic (intramuscular benzylpenicillin and gentamicin are options for infants under 2 months of age).
- Supportive measures such as continued feeding to avoid malnutrition, antipyretics to reduce high fever and protection from cold (especially keeping young infants warm) are part of the management.
- Proper advice is given to caretakers of non-severe cases on home care, including compliance with antibiotic treatment instructions.
- Signs of malnutrition are assessed as this increases the risk of death due to pneumonia. Severely malnourished children are referred to hospital.

3.17.10 Management of the disease

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Pneumonia can be treated with antibiotics. These are usually prescribed at a health centre or hospital, but the vast majority of cases of childhood pneumonia can be administered effectively within the home. Hospitalization is recommended in infants aged two months and younger, and also in very severe cases.
3.18 Toxoplasmosis

ICD-10 B58

3.18.1 Identification

A parasitic disease caused by the protozoan *Toxoplasma gondii*. The parasite infects most commonly warm-blooded animals, including humans, but the primary host is the feline (cat) family. Animals are infected by eating infected meat, or by transmission from mother to fetus. Although cats are often blamed for spreading toxoplasmosis, contact with raw meat is a more significant source of human infections in many countries.

**Clinical features:**
- 80% of cases are asymptomatic.
- Symptomatic patient has enlarged lymph nodes, especially around the neck, muscle pain, intermittent fever and malaise.
- Repeated abortion in child baring women.
- In early pregnancy brain damage, liver and spleen and eye may occur.
- In late pregnancy may result in persistent eye infection through life.
- Toxoplasmosis acquired after birth usually result in no symptoms or only mild illness.

**Diagnosis:** Biopsy, Serological test (IFAT, ELASA)

3.18.2 Infectious agent

Protozoan *Toxoplasma gondii*

3.18.3 Occurrence

**Situation analysis in Iraq of Toxoplasmosis from 2000 – 2009**
The disease is reported in all provinces in Iraq, affect mainly females. High numbers of cases were reported in Erbil (373), Basrah (102), Baghdad Rusafa (55), and Diyala (35) in 2009.

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<tr>
<th>Year</th>
<th>2000</th>
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<th>2004</th>
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<td>643</td>
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</tbody>
</table>

3.18.4 Reservoir

Main host is cats; intermediate hosts include sheep and goats.

3.18.5 Mode of transmission

- Adult most acquire toxoplasmosis by eating raw meat or undercooked meat infected with tissue cyst.
- Children may become infected by ingestion of oocytes in dirt or sand pit after fecal contamination by cats, particularly kitten or other animals.
3.18.6 Incubation period

5-23 days

3.18.7 Period of communicability

Toxoplasmosis is not passed from person-to-person, except in instances of mother-to-child (congenital) transmission and blood transfusion or organ transplantation. Kittens and cats can shed millions of oocysts in their feces for as long as 3 weeks after infection. Oocysts shed become infective from 1-5 days later and can remain infective in moist soil or water for over a year. Additionally, oocysts can remain infective in the meat of an infected animal until it is thoroughly cooked.

3.18.8 Susceptibility and resistance

General. Asymptomatic pregnant woman can transmit infection to her unborn child. Infection is often highest in areas of the world that have hot, humid climates and lower altitudes.

3.18.9 Methods of control

Increase health education of child bearing women and advise them to:

- Cook meat thoroughly and avoid uncooked meat.
- Wear gloves during gardening and wash hands thoroughly after work and before eating.
- Control stray cats.

3.18.10 Management of the disease

- Those without symptoms typically do not need treatment.
- Medications to treat the infection include an antimalarial drug and antibiotics. AIDS patients should continue treatment for as long as their immune system is weak to prevent the disease from reactivating.

---

3.19 Typhoid and Paratyphoid fever

ICD-10 A01.0: Typhoid Fever
ICD-10 A01.1-A01.4: Paratyphoid Fever

3.19.1 Identification

A systemic bacterial disease with insidious onset of sustained fever, severe headache, malaise, anorexia, relative bradycardia, nonproductive cough in early stage of illness, rose spots on the trunk and constipation more often than diarrhea in adults. Intestinal hemorrhage or perforation can occur in 1% of cases. Case fatality rate is 10%-20% without antibiotic therapy and 1% with antibiotic use. Relapse may occur in 15%-20% of patients, but with milder form.

Paratyphoid fever presents a similar clinical picture, but tends to be milder, and the case-fatality rate is much lower.

Case classification:
- **Suspected case:** Any case having the following features: sustained, non-sweating fever of 38 °C or more, for 3 days or more, abdominal discomfort (abdominal pain, diarrhea or constipation). With 2 or more of the following symptoms: dry non-productive cough, relative bradycardia, anorexia, severe headache.
- **Confirmed Case:** A suspect or probable case with detection of *S. typhi* or *S. paratyphi* through positive culture of blood, stool, urine or bone marrow (laboratory investigation: culture of blood early in the disease; stool and urine after the first week; or bone marrow culture which provide the best bacteriologic confirmation (90%-95% recovery) even in patients who have already received antimicrobials. Because of its limited sensitivity and specificity, serologic tests (widal test) are generally of little diagnostic value.)
- **Carrier:** any person discharging bacilli in stool or urine for more than a year following infection.

3.19.2 Infectious agent

- For typhoid fever: *Salmonella typhi*
- For paratyphoid fever: *Salmonella paratyphi A*

3.19.3 Occurrence

Worldwide, mostly endemic in many developing countries, especially in the Middle East. It occurs throughout the year with seasonal increase in summer months.

3.19.4 Reservoir

Humans, rarely domestic animals for paratyphoid.
3.19.5 Mode of transmission

Ingestion of food and water contaminated by feces and urine of patients and carriers.

3.19.6 Incubation period

Depends on inoculum size and host factors; from 3 days to over 60 days (range 8-14 days). For paratyphoid is 1-10 days.

3.19.7 Period of communicability

As long as bacilli appear in excreta, from first week throughout convalescence (1-2 weeks for paratyphoid).

3.19.8 Susceptibility and resistance

General and is increased in individuals with gastric achlorhydria and possibly in those who are HIV- positive. Relative specific immunity follows recovery from clinical disease, inapparent infection and active immunization. In endemic areas, typhoid fever is most common in preschool children and children ages 5-19 years old.

3.19.9 Methods of control

3.19.9a Preventive measures

1) Educate the community about the importance of hand washing.
2) Dispose human feces in a sanitary manner.
3) Protect, purify and chlorinate public water supply.
4) Control fly by screening, spraying with insecticides; control fly breeding by frequent collection and disposal of garbage.
5) Clean preparation and handling of food.
6) Pasteurize or boil all milk and dairy products.
7) Good personal hygiene of patient, convalescent and carriers.
8) Encourage breast-feeding throughout infancy; boil all milk and water used for infant feeding.
9) Periodic examination of the food handlers and exclusion of chronic carriers from work until three consecutive negative stool cultures are obtained (and urine at areas endemic with schistosomiasis) at least one month apart (for acute cases 24 hour apart).
10) Immunization of the high risk group.

3.19.9b Control measures

1) Suspected cases should be reported from all health care facilities to higher level.
2) Confirmed cases should be investigated using case investigation form; enteric precautions for acute cases and should be supervised until 3 consecutive negative cultures of feces (and urine in patients with schistosomiasis) at least 24 hours apart.
and at least 48 hours after any antimicrobials, and not earlier than 1 month after onset. If any of these is positive, repeat cultures at monthly intervals during the 12 months following onset until at least 3 consecutive negative cultures are obtained.

3) Search for unreported cases, carriers or contaminated food, water, milk or shellfish.
4) Household and close contacts should not be employed in sensitive occupations (food handlers) until at least 2 negative feces and urine cultures, taken at least 24 hours apart, have been obtained.

3.19.9c Epidemic measures
Search for source of infection (case or carrier) and for the vehicle (water or food) through which infection was transmitted. Elimination of suspected contaminated food. Water supplies should be chlorinated. Vaccination before or during an outbreak give protective efficacy of 72%.

3.19.9d Disaster implications
Restoration of safe drinking-water supplies and excreta disposal facilities. Selective immunization of stabilized groups such as school children, prisoners, and municipal or hospital personnel may be helpful.

3.19.9e International measures
Immunization is not required for entry to any country. Immunization is advised for international travellers to endemic areas, especially if exposed to unsafe food and water, or close contact in rural areas to indigenous populations.

3.19.10 Management of the disease
Suspected cases of typhoid should be treated with broad-spectrum empiric antibiotics immediately. Treatment should not be delayed for confirmatory tests since prompt treatment drastically reduces the risk of complications and fatalities. Antibiotic therapy should be narrowed once more information is available.

Compliant patients with uncomplicated disease may be treated on an outpatient basis. They must be advised to use strict hand washing techniques and to avoid preparing food for others during the illness course. Hospitalized patients should be placed in contact isolation during the acute phase of the infection. Feces and urine must be disposed of safely.

---

## Annex 1: Communicable Diseases notified in 2011 in comparison with 2010

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### Annex 2: Toolkit for the Important Vectors and Vector-borne Diseases in Iraq

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حمى الوادي المتصدع  
السحايا الدماغية  
الفلاريا الليمناوية (داء الفيل) |
| 3   | بعرض الايدس | حمى الضنك  
حمى الصفراء  
حمى الثلاثة ايام (حمى البيناسي)  
امراض الليمانيات (حمى بغداد والكلازازر) |
| 4   | ذباب الرمل | التايفوين والباراتايفونيد  
daيزنتري  
سببات الكوليرا  
سببات التسمم الغذائي  
امراض العين (الرمد الصدري)  
الحمى القمحية أو الجمرة الخبيثة  
بيوض بعض الديدان مثل الإسكاس والدودة الوحيدة  
بعض الرشحات مثل رشح شلل الأطفال ورشح الجدري |
| 5   | الدبابة المنزلي | التدويد / برقاتها تهاجم الجروح والقروح والدمام  
الذبابة الحزازونية  
الذبابة السوداء  |
<p>| 6   | ديدان الفلاريا / عمي الانهار |     |</p>
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## Annex 3: Toolkit for the Pesticides used in the field of Public Health

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المادة الأولى:
تُحدد الأمراض الانتقالية في المجموعات التالية

- المجموعة الأولى
  - الكوليرا
  - الشلل الرخوي الحاد وشلل الأطفال
  - متلازمة العوز المناعي المكتسب (الإيدز)
  - الحمى النزفية
  - الملاريا الخبيثة
  - الخناق
  - الحصبة
  - التسمم={(الشيقي)}
  - الزحار

- المجموعة الثانية
  -得天ال السحائي السحائي
  - الطاعون
  - الحمى الصفراء
  - الحمى الراجعة
  - الجمرة الخبيثة الرنة
  - التيفوس
  - الحصبة الألمانية

- المجموعة الثالثة
  - داء الكلب
  - السعال الديكي
  - الشلل الرخوي المزمن
  - الالتهاب الليفي (الحمى السوداء)

- المجموعة الثالثة
  - الزحار
  - السعال الديكي
  - الرئة
  - الملاريا الخبيثة
  - التسمم
  - الكوليرا
  - الشلل الرخوي الحاد وشلل الأطفال
  - متلازمة العوز المناعي المكتسب (الإيدز)
  - الحمى النزفية
  - الملاريا الخبيثة
  - الخناق
  - الحصبة
  - التسمم={(الشيقي)}
  - الزحار

تعليمات رقم 1 لسنة 2007

استنادًا إلى أحكام المادة 45 من قانون الصحة العامة رقم 89 لسنة 1981 أصدرنا التعليمات الآتية

- المجموعة الأولى
- المجموعة الثانية
- المجموعة الثالثة
الالتهاب السحائي الفيروسي
التهاب السحائي الجرثومي
الكلاف
التدرين الرئوي
التدرين خارج الرئة
الأمراض المنقلة جنسيا
الجرب
الحميدة
الرئة
الحميدة
الملاريا الحميدة
الأمراض المنقلة جنسيا
الجرب
الحميدة
المخالب
الجلدية الخبيثة
الكلاف
الأمراض المنقلة جنسيا
الحصبة
الجرب
الحميدة
الملاريا
الحميدة
الأمراض المنقلة جنسيا

التعليمات

أولاً - على دوفتر المهن الطبية والصحية العامين في الممرضات الرسمية الإخبار فورًا وبأي وسيلة اتصال متاحة
ولخلال (24) أربع وعشرين ساعة بالجهة الصحية العليا من اكتشاف أي مرض من أمراض المجموعة الأولى
منصوص عليها في البند (أولا) من المادة (1) من هذه التعليمات.
ثانيًا - على دوفتر المهن الطبية والصحية العامين في القطاع الخاص الإخبار فورًا وبأي وسيلة اتصال متاحة
ولخلال (24) أربع وعشرين ساعة بالجهة الصحية العليا من اكتشاف أي مرض من أمراض المجموعة الأولى
منصوص عليها في المادة (1) من هذه التعليمات.
ثالثًا - على كل شخص علم بوجود أي مرض من أمراض المجموعة الثانية (الثانية) الإخبار عنها خلال (7) سبعة أيام
ولخلال (30) ثلاثون يومًا عن أمراض المجموعة الثالثة المنصوص عليها في البندين (ثانيا و (ثالثا) من المادة (1)
من هذه التعليمات.
رابعة - عند نفاذ الروتين في أحد الأمراض المنصوص عليها في البندين (ثانيا و (ثالثا) من المادة (1) من هذه التعليمات أو
ظهور حالات مرضية غير معروفة أو غير مسجلة فيلزم أخبار الجهات الصحية المنصوص عليها في البند (أولا)
من هذه المادة.

العيساوي
راعي الصحة وكالة
伍ط卿
العراقية
الرابعة

المواد والمعاهد

يعاقب المخالف لأحكام المادة (2) من هذه التعليمات وفقاً للمادة (96) من قانون الصحة العامة رقم 89 لسنة 1981

المادة الرابعة

تلقى تعليمات تحديد الأمراض الانتقالية رقم 3 لسنة 1997

المادة الخامسة

تنفذ هذه التعليمات من تاريخ نشرها في الجريدة الرسمية

درافت حيد العيساوي
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2007/6/17
References

The Geographic data of the PHCs clinics are currently being updated and verified by MOH (September, 2011).