A HANDBOOK OF EMERGENCIES

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Eighth Edition

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Preface

Our apologies for the undue delay in bringing out the new revised edition of this popular pocketbook. The delay was largely due to a handover to the new accomplished publisher M/s Jaypee Brothers Medical Publisher (P) Ltd, New Delhi, India.

The text, as before, covers all aspects, though somewhat in brief, of common emergencies encountered in a day-to-day practice.

Our special thanks to Dr J Lalkaka, Dr Amit Desai and Dr Rumi Jehangir for their inputs.

Aspi F Golwalla
Sharukh A Golwalla

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Emergencies in Tropical and Infectious Diseases

Malaria

Morbidity and mortality from malaria are increasing owing to the increasing drug resistance and lack of effective methods to control prevalence in those areas where transmission is most intense. The infection can pose different problems depending on the species of the parasite. Falciparum malaria in an adult is a multisystem disorder. A thick blood film establishes the diagnosis.

Manifestations of Complicated, Severe Malaria

1. *Encephalopathy:* Altered consciousness, coma, convulsions (cerebral malaria). Coma may be moderate or profound, and may be accompanied by hypertonicity, opisthotonus, conjugate deviation of gaze, or by generalized hypotonia. Convulsions may be focal or generalised, brief or protracted.
2. *Acute renal failure* is caused by acute tubular necrosis, and is therefore reversible if patient can be kept alive.
3. *Severe anaemia* may be present at time of diagnosis, or may develop rapidly during early stages of treatment.
4. *Adult respiratory distress syndrome* has a poor prognosis.
5. *Pulmonary oedema* is usually caused by excessive fluid input in a patient with renal impairment or failure.

6. *Hyperlacticacidemia* is caused by impaired tissue perfusion in tissues occupied by sequestered parasites, and is exacerbated by dehydration and hypoglycemia.
7. *Hypoglycemia* occurs especially in pregnancy. It is caused by impaired hepatic gluconeogenesis. It may also result from quinine therapy, when the stimulatory effect of the drug on pancreatic beta cells raises plasma insulin concentration.
8. *Intravascular hemolysis* leads to hemoglobinemia and hemoglobinuria.
9. *Jaundice,* which is seldom deep, is a combination of hemolysis and impaired hepatic function.
10. *Disseminate intravascular coagulation* is occasionally severe enough to cause overt bleeding.
11. *Shock* may be a feature of severe anemia. In individuals with indwelling venous or urethral catheters it may be caused or accompanied by bacteremia.
12. *High fever* and hyperpyrexia.

Management

1. *Hospitalization* in ICU.
2. *Pass nasogastric* tube if pt semiconscious or in coma.
3. *Fluids IV* according to urine output and JVP. Excess of fluids should be avoided because of danger of pulmonary oedema.
4. *Antimalarial drugs* (i) Artesunate monotherapy 4 mg/kg loading dose on first day followed by 2 mg/kg od × 6 days. Severe malaria 2.4 mg/kg IM, followed by 1.2 mg/kg at 12 and 24 h daily, then 1.2 mg/kg daily for 6 day; or 2.4 mg/kg IV on first day followed by 1.2 mg/kg daily until patient can take orally or another effective antimalarial drug. (ii) Artemether—Severe malaria 3.2 mg/kg im as loading dose on first day followed by 1.6 mg/kg daily for minimum of 3 days until patient

can take oral therapy to complete a 7 day course. (iii) Artemether (120 mg) and lumefantrine (120 mg). Dose: Standard 3 days treatment schedule with total of 6 doses as follows 4 tablets as a single dose at time of diagnosis, again 4 tablets after 8 hours and then 4 tablets bd for 2 days (total 24 tablets). (iv) Quinine sulphate 20 mg/kg in 5% saline as IV infusion over 4 hours till parasites clear, then 8 hourly till patient can takes oral medication. (v) Arterolane maleate 150 mg and piperaquine phosphate 750 mg one tablet daily for 3 days.

Management of Complications

a. *ARF:* Fluid restriction, reduction of maintenance dose of antimalarial drug by one third, and daily measurements of creatinine and electrolytes. Peritoneal dialysis or hemodialysis.
b. *Anemia:* Blood transfusion. Exchange transfusion using packed cells may be required.
c. *Hypotension and shock:* Volume replacement and dopamine.
d. *Hpoglycemia:* 5–10% dextrose IV.
e. *Convulsions:* Diazepam.
f. *Hyperpyrexia:* Tepid water or alcohol sponging, covering with wet sheet and fanning, paracetamol.
g. *Pulmonary oedema:* Oxygen, frusemide IV and positive pressure ventilation.
h. *ARDS or shock:* Ventilatory support.
i. *Associated bacterial infection:* Pneumonia, gram-negative bacteremia, urinary infection. Treatment with appropriate antibiotics.
j. *Coagulopathy:* Fresh frozen plasma, or platelet infusion. DIC may be caused by gram-negative sepsis which should be covered by antibiotics.

Management of malaria in pregnancy: High degrees of pyrexia can initiate premature labour, hence temprature must be lowered promptly. Chloroquine can be prescribed. Quinine is safe and in the usual dosage does not produce uterine stimulation. Arterolane 150 mg and piperaquine phosphate 750 mg tablet one od × 3 days is safe and clears parasitemia more effectively than quinine. Exchange tranfusion if heavy parasitemia (>20% parasitized cells). It also allows replacement of RBCs and clotting factors without volume overload.

Typhoid Fever

Salmonella infection can cause typhoid (and paratyphoid) fevers, a bacteremic illness with systemic manifestations. Fulminant states may be due to heavy infection or delayed diagnosis.

Clinical Features

Clinical features are those of septicemic state. Patients is toxic with high fever and a delirious confusional state sets in. Marked abdominal distension with 'pea soup' stools. Hemorrhage and perforation are likely to occur. Toxic myocarditis is another serious complication. Hypotension, shock, a persistent lactic acidosis and acute renal failure may result in death.

Diagnosis

S. typhosus can be grown by blood or bone marrow culture. Blood culture is the usual diagnostic method. Widal test lacks specificity and sensitivity.

Management

(1) Hospitalization. (2) Correction of dehydration with IV fluids. (3) Inotropic support. (4) Antibiotics—Ofloxacin

500 mg bd for 10 days and/or ceftriaxone 2 g IV daily for 10 days. Chloramphenicol 500 mg IV q6h, then 500 mg q8h for 10 days may be tried. Levofloxacin 250 gm BD PO, cefotaxime 500 mg IM BD.

Fulminant Amoebic Infection

Amoebiasis is a common infection of the GI tract. Manifestations which present as emergencies are severe acute amoebic dysentery and liver abscess or abscesses from blood-borne dissemination of amoebae.

Fulminating amoebic colitis is characterised by numerous bloody stools (20 or more/day), generalised abdominal discomfort, colicky pain preceding evacuation, and rectal tenesmus, which tends to be constant and intense. Advanced lesions may cause GI hemorrhage or perforation with peritonitis. Fulminating amoebic colitis may be encountered in pregnancy, malnourished, immunocompromised individuals and those receiving corticosteroids for erroneous diagnosis of ulcerative colitis.

Amoebomas usually have an acute evolution with bloody diarrhoea or dysentery of moderate severity, abdominal pain and a palpable mass in the colonic area.

Acute appendicitis has clinical features similar to those of bacterial appendicitis, but in many cases, there is no involvement of the caecum, giving rise to bloodstained diarrhoea.

Diagnosis

(a) Stools may reveal trophozoites with ingested RBCs. (b) Rectosigmoidoscopy and immediate microscopic examination of rectal smears will reveal presence of motile, haematophagus trophozoites. (c) Serology—Anti-amoebic antibodies can be detected in more than 75% of pts with colonic invasive amoebiasis.

Management

(a) Correction of dehydration. (b) Metronidazole 500 mg IV q8h for 10 days. (c) Tetracycline 250 mg qds for 10 days.

Fulminating colitis and amoebic appendicitis require surgical treatment in addition to chemotherapy. In fulminating colitis, only partial or complete resection of the colon offers some hope of recovery.

Amoebic liver abscess is more common in adult males excessive alcohol and malnutrition are predisposing factors. The abscess is usually single, and the most common location right lobe of the liver.

Clinical Features

In most patients there is an abrupt onset with pain in the region of the liver, which is intense and constant, and radiates to the scapular region and right shoulder. Local oedema of chest and abdominal wall and signs of effusion nay be detected. Fever is common. Nausea and vomiting may occur.

Complications

Liver abscesses may heal following, adequate chemotherapy, increase in size, rupture or disseminate. Rupture may occur into—(a) Peritoneum presenting as acute abdomen. (b) Pericardium. A left lobe abscess may extend into the pericardium. (c) Into pleural space causing empyema, or into lung giving rise to lung abscess. (d) Retroperitoneal into posterior abdominal wall with swelling over kidney region. (e) Right paracolic gutter with tenderness in rt. iliac fossa. (f) Into subphrenic space giving rise to subphrenic abscess.

Diagnosis

(a) Imaging—CXR, ultrasound or CT scan of abdomen. (b) Serology—Anti-amoebic antibodies can be detected in serum of 95% of patients.

Management

1. *Chemotherapy:* Metronidazole or tinidazole as for amoebic dysentery.
2. *Percutaneous aspiration:* Preferably under CT guide. Indications - Left lobe abscess, massive abscess, a palpable mass, persistent localized tenderness, markedly raised hemidiaphragm, pleurtitic pain suggesting an impending leak, failure of symptoms to remit on drug therapy.

Technique: The needle is introduced into area of maximum tenderness or into 8th or 9th intercostal space in midaxillary line. All available pus should be removed. If abscess is on lower surface of left lobe, aspiration should be performed through an open abdomen.

If perforation—Gastric suction, IV fluids, and electrolytes together with Ceftriaxone 1g daily and metronidazole IV. Surgery should be avoided.

Acute Bacillary Dysentery

The passage of blood and pus cells associated with infective diarrhoea constitutes acute dysentery. Infection with species of *Shigella*. *Sh. dysenteriae* can produce fulminating illness.

Clinical Features

In more severe forms of the disease, there is acute phase response with high fever, anorexia, headache and malaise with frequent passage of small volumes of stool, progressing to passage of frank blood and pus.

Complications

(1) Bacteremia can occur in immunocompromised subject. (2) Hemolytic-uremic syndrome consisting of

microangiopathic hemolytic anemia, thrombocytopenia and renal failure.

Diagnosis

It is made by isolating the organism from the stool using a selective medium.

Management

(a) Hospitalization. (b) IV fluids and electrolytes to correct dehydration. (c) Antibiotics—ciprofloxacin 500 mg qds and co-trimoxazole 2 tablets BD. Antimotility drugs should be avoided.

Cholera

Cholera is usually the result of infection with strains of *Vibrio cholerae*. Infection is confined exclusively to the lumen of GI tract where a powerful enterotoxin is formed, which stimulates secretion of isotonic fluid. Morbidity and mortality of the disease is attributable to the consequences of dehydration.

Clinical Features

Watery diarrhoea starts abruptly and soon the stools acquire a 'rice water' appearance. Vomiting is common. Within a few hours the patient becomes rapidly dehydrated. A state of shock may ensue. Fever is uncommon, but severe muscle cramps do occur.

Diagnosis

Bacterial confirmation is not necessary during epidemics. *V. cholerae* can be cultured from stool or rectal swab.

Management

1. *Management of diarrhoea:* A cholera bed can be fashioned from a canvas stretcher by cutting in the centre a hole through which liquid stool drains directly into a bucket beneath. Thus fluid losses can be easily measured, or use of bedpan, or absorbent pads if stools numerous.
2. *Oral rehydration solution:* Constituents (g/L)

Sodium chloride	3.5
Sodium bicarbonate	2.5
Potassium chloride	1.5
Glucose	2.0

   Small volumes of ORS should be given frequently with cup or glass or if necessary through nasogastric tube. If the above salts are not available, an appropriate solution can be prepared by mixing 5 g of household salt with 40 g sucrose (or 20 g glucose) in 1 litre of water. Depending on the amount or extent of dehydration and ongoing losses, pts may require 100–300 mL/kg/day.
3. *IV rehydration* should be reserved for those who are much dehydrated, shocked or vomiting so severely that oral rehydration is impossible. Solution containing sodium chloride 5 g, sodium bicarbonate 4 g and potassium chloride 1 g in distilled water. Or 2 litres of isotonic saline can be alternated with 500 mL 1/6 molar sodium lactate or isotonic sodium bicarbonate.

   Access to circulation may be difficult to maintain in shocked, dehydrated patients, and central venous cannulation may be necessary. Sometimes even this is not possible, alternative approaches such as

intraperitoneal or subcutaneous infusion may be required. Initial infusion should be rapid until pulse and BP have been restored.

4. *Antibiotics:* One of the following can be prescribed—(a) Tetracycline 50 mg/kg/day in 4 divided doses for 3 days. (b) Ampicillin 500 mg q6h can be given IV. Appropriate drug in pregnancy. (c) Doxycycline 300 mg as single dose. (d) Ciprofloxacin 500 mg bd for 5 days.

Acute Gastroenteritis (Food Poisoning)

Severe food or water-borne infection may present as an urgency.

1. *Acute staphylococcal food poisoning* occurs after ingestion of food which has been improperly cooked or stored. Although it may be severe and prostrating, it is usually short-lived.
2. *Salmonellae food poisoning* comes from ingestion of food (e.g. meat, eggs) or dairy products. Host factors (extremes of age, debilitating disease, immunosuppression, gastric hypoacidity), increase susceptibility to severe illness and bacteremia.
3. *Seafood poisoning* results from algae-produced toxins and also from poisoning caused by eating contaminated fish or prawns. Without emergency treatment, the victim can suffer respiratory failure.

Clinical Features

Acute enterocolitis manifests itself in severe illness as passage of voluminous, watery stools every half hour or more frequently leading to rapid dehydration and renal insufficiency. The illness begins with nausea and vomiting,

abdominal cramps often with vomiting, chills and fever. Later the volume may decrease and blood and mucus appear. Abdominal tenderness is common.

Management

(a) IV fluids and electrolytes. (b) Antibiotics—Ciprofloxacin 500 mg BD PO or 200 mg IV for 5 days.

Campylobacter Enterocolitis: Infection occurs by consumption of contaminated beef, under cooked poultry, or milk.

Clinical Features

Typical symptoms begin with headache, malaise, backache, myalgia and pyrexia of about to 40°C. A few hours later severe abdominal cramps and diarrhoea begin. The stools may be loose or watery or mainly bloody. Nausea is common but vomiting rare.

Diagnosis

Isolation of the organism from stool. Blood culture may be positive in the immunocompromised host.

Management

(1) Fluid and electrolyte replacement. (2) Antibiotics—Ofloxacin 400 mg q6h or Ciprofloxacin 500 mg BD.

Clostridium Difficile Infection

Risk factors: Gastric acid suppression with proton pump inhibitors, enteral feeding, GI surgery, cancer chemotherapy, immunosuppression with corticosteroids and haemopoietic stem cell transplantation.

Diagnosis

(a) Presence of diarrhoea—Passage of 3 or more stools in 24 hours or fewer consecutive hours. (b) Stool test positive for presence of toxigenic *C.difficile* or its toxins or colonoscopic or histopathologic findings demonstrating pseudomembranous colitis.

Treatment

(i) Oral rehydration therapy. (ii) Single dose octreotide 100 mg stat as antisecretory agent together with antibiotics.

Tetanus

Tetanus is caused by a powerful neurotoxin produced by strains of *Clostridium tetani* when introduced into the tissues. Portals of entry are contamination of wounds by manure or rusty instruments, bums, otitis media, septic abortions, child birth, surgery, animal bites and stings.

Clinical Features

1. *Rigidity:* Spasm of masseters produces lock jaw (trismus) and of face muscles, risus sardonicus. Spasm of erector spinae and of abdominal muscle gives rise to exaggerated lumbar lordosis, neck retraction and abdominal rigidity.
2. *Muscle spasms* occur in severe tetanus. Seizures can be induced by external stimuli. Spontaneous seizures may result in continuous convulsive state. Spasm of pharynx causes dysphagia and of larynx can lead to asphyxia. Patient may be hypoxic and cyanosed due to spasm of respiratory ms.
3. *Symptoms due to sympathetic overactivity:* Tachycardia, cardiac arrhythmias, fluctuating BP, fever, excessive sweating and salivation.

Featuresof fulminant tetanus are generalised spasticity with prolonged spasms, tachypnoea (>40 breaths/min), apnoeic spells, marked tachycardia and severe hypertension and tachycardia alternating with bradycardia and hypotension, or severe persistent hypertension or hypotension.

Complications

1. *Respiratory:* (a) Bronchopneumonia can result from aspiration of stomach contents, blockage of airways by sticky secretions and lung collapse. (b) Asphyxia. (c) Respiratory fixation - Chest fixation in which general hypertonicity involves pectoral, intercostal and ms. of abdominal wall reducing vital capacity markedly.
2. *Due to spasms*: Muscle tear and wedge fracture of thoracic vertebrae.
3. *Autonomic instability:* Death may occur from ventricular arrhythmia or from profound shock.
4. *Miscellaneous:* Hyperpyrexia, dehydration, paralytic ileus, development of catabolic state and adverse effects of drugs.

Management

1. *Admission into ICU.*
2. *Mechanical support* if neuromuscular blockade is required to control muscle spasms and impaired respiration. If hyperventilation occurs danger of aspiration secondary to gastric tube feeding. Bladder catheterization if urinary retention, frequent turning and chest physiotherapy.
3. *Wound care:* Thorough debridement especially if deep puncture wound.
4. *Antitoxin* to neutralise free toxin for adults human tetanus immunoglobulin 3000 units IM given at

separate sites. If horse serum tetanus antitoxin has to be used dose's 50,000 U IM or IV (risk of serum sickness).

5. *Sedatives and muscle relaxants:* Diazepam dose depends on severity. In a severe case 10–20 mg IV q3h. For less severe cases 5–10 mg PO 92 hours. Midozolam. Adults 0.1 to 0.3 mg/kg in IV infusion is preferred for prolonged therapy.
6. *Antibiotics:* Doxycycline 100 mg PO BD and metronidazole 500 mg PO q8h.
7. *Tracheostomy and ventilation* should be undertaken early in patients whose disease is not controlled with conservative sedative regimen, because inadequate control of muscle spasms results in asphyxia and depression of swallowing reflex.
8. *Induced neuromuscular paralysis:* Pancuronium 2–4 mg every 1/2-1 hr by bolus IV injection or continuous IV infusion to have a well regulated subtotal paralysis for efficient ventilatory support.
9. *Management of autonomic circulatory disturbances*: (a) Hypotensive spells—Volume load or if ineffective dopamine. (b) Hypertensive spells (Refer). (c) Bradycardia—IV atropine. (d) Sustained tachycardia—Verapamil 40 mg TDS PO.
10. *Nutrition:* Adequate calories with 75–100 g protein through nasogastric tube.
11. *Care of the wound:* Removal of foreign material and debridement of non-viable tissue of entry wound. Tetanus toxoid injection.

Other clinical types which can be severe are: (a) Cephalic tetanus results from injuries to head and neck and infections of eye and orbit. One or more cranial nerves mostly Vth may be involved with spasm and

paralysis of muscles they supply. Cephalic tetanus can evolve into generalised form. (b) Neonatal tetanus due to sepsis of the umbilical stump carries a high mortality. Typically the week-old infant presents with inability to suck because of spasm of facial, pharyngeal muscles, or with generalised convulsions. Crying is hoarse and the face screwed up. Cyanosis and apnoea accompany the more severe spasms.

Leptospirosis

Infected rodents are typically the source of human infection which are most commonly due to contact with water or environmental surface contaminated with infected urine. Risk of leptospirosis is especially during rainy season.

Clinical Features

1. *Anicteric leprospirosis:* (a) Septicemic stage: Abrupt onset of fever, chills and rigors, persistent headache, photophobia, conjunctival suffusion, cough, etc. (b) Immune stage: Most of the symptoms return after 1–3 days period of minimal symptoms but are decreased. In addition aseptic meningitis is the hallmark of the immune stage.
2. *Icteric leptospiriosis* (Weil's syndrome): Liver enlarged and tender with increasing severity of jaundice, the patient is at greater risk of developing renal failure, hemorrhage and cardiovascular collapse.

Diagnosis

Isolation of the pathogenic leptospira is the only definite proof of infection, though serological methods can be used.

Management

(a) Hospitalization in ICU in patients with severe infection and Weil's disease. (b) Antibiotics—Ampicillin 500–1000 mg IV q6h. In less severe cases amoxycillin 500 mg PO q6h for 5–7 days or Doxycycline 100 mg PO BD. (c) Doxycycline if penicillin allergy 200 mg PO QDS or tetracycline 500 mg initial, then 250 mg qds × 6d or ceftriaxone 1 g OD × 7d.

Dengue

Diagnostic Criteria

Dengue hemorrhagic fever

1. Fever for 2 or more days.
2. Thrombocytopenia ($<100 \times 10^6$/L).
3. Capillary leakage, pleural effusion on right side, rising hematocrit (usually more than 20% higher).
4. Evidence of recent dengue infection.

Dengue shock syndrome only: In addition to above, hypotension for age, or narrow pulse pressure (± 20 mm Hg).

Clinical Features

1. Occurs predominantly in children under 14 years of age.
2. Febrile phase is unremarkable.
3. With defervescence, there is sudden onset of weakness, cool extremities, agitation, mild-epigastric abdominal pain, petechiae, circumoral cyanosis, thrombocytopenia, capillary leakage and hypotension.
4. Hypovolemia.
5. Hepatomegaly within 1–2 days.
6. Post-illness bradycardia.

Investigations

Platelet count, hematocrit, chest X-ray, and ultrasonography or other techniques to detect ascites and pleural or pericardial effusions. Also measurement of bleeding time, prothrombin time, liver enzymes and serum complement. Etiology can be established by recovery of the virus from the acute phase serum, usually on or before 5th day of onset of fever.

Management

1. Immediate evaluation of vital signs, hemoconcentration, dehydration, urine output and electrolyte imbalance.
2. O_2 if laboured breathing or cyanosis.
3. Rapid IV replacement of fluids and electrolytes. Care should be taken to avoid overhydration (fall in hematocrit and wide pulse pressure).
4. Fresh frozen plasma and platelets or heparin (if laboratory evidence of severe consumptive coagulopathy), together with vigorous replacement of fluid and protein with plasma or colloids.

Note: If patient is bleeding, it is important to distinguish whether this is a complication of dengue fever or a manifestation of DHF/DDS. In dengue fever with severe bleeding, the hematocrit decreases and evidence for increased vascular permability (ascites or pleural effusions) is absent.

Chickungunya Fever

Clinical Features

Although rare in adults, children, particularly neonates can develop vomiting and/or diarrhoea and meningio-encephalitis.

Neurological manifestations such as encephalitis, febrile seizures, meningeal syndrome and acute encephalopathy have been observed, severe dehydration can occur.

Management

At home

1. Adequate rest, heat may increase/worsen joint pains.
2. Paracetamol tablets 500 mg qds, children 50–60 μg/kg body weight per day in divided doses.

Hospitalization if:

1. Fever persisting for >5 days.
2. Intractable pain.
3. Postural dizziness, cold extremities.
4. Decreased urine output.
5. Incessant vomiting.

All patients must be assessed for dehydration and proper rehydration therapy (preferably oral) to be started quickly. Severe dehydration is characterized by two of the following:

- Abnormal sensorium, excessive sleepiness or lethargy
- Sunken eyes
- Poor fluid intake
- Dry parched tongue
- Reduced skin turgor (very slow skin pinch taking 2 sec to retract).

Effects of Heat

Clinical manifestations needing emergency attention:

1. **Heat syncope:** A fainting spell or 'blackout' from excessive heat and humidity. The individual drops to the floor if standing and there is transient loss of consciousness. *Treatment:* Laying down the person flat in cool surroundings is followed by quick recovery.

2. **Heat stroke** (Sunstroke, heat hyperpyrexia) is characterised by sudden loss of consciousness which may be preceded by prodromal signs of cerebral irritation namely headache, dizziness, nausea, convulsions and visual disturbances. There is very high fever (105–107°F) and cessation of sweating. The skin is flushed and dry, pulse rapid, irregular and weak and hypotension.
   Management: (a) Cooling by fanning after sprinkling water, immersion in cold water or use of ice packs to neck, axillae and groins, or ice water enemas. (b) Massage of extremities to maintain circulation. (c) Normal saline 1000 mL infusion if dehydration or cramps. (d) Do not give antipyretics like paracetamol, aspirin.
3. **Exertional heat injury** occurs in those who indulge in physical exertion in hot and humid envionment. Patients suffer from headache, nausea, incoherent speech and muscular incoordination. Body temperature is increased with sweating. There is tachycardia and hypotension.
   Treatment: (1) Patient should be nursed in cool envrironment and covered with weight cold sheets. (2) IV Dextrose saline infusion. (3) Massage of extremities, helps in increasing peripheral blood flow.

Rabies

Since rabies is a fatal infection, it is solely of academic interest.

Measles

In developing countries, measles is a major cause of childhood morbidity and mortality since a large number of children are not vaccinated. It is also a significant factor

in the development of—(a) Protein-energy malnutrition which contributes to a large number of deaths, and (b) peumonia which can be fatal in young children, and (c) Vitamin A deficiency which can increase child mortality.

Complications

1. *Pneumonia* is a common complication and the main cause of death. It is caused by the measles virus itself, the peumonia usually occurs in the first 2 weeks of the illness. It is often however the result of superinfection by other viruses or bacteria, including *Staph. aureus* or Gram-negative organisms. During the latter part of the exanthematous stage, herpes simplex virus and adenoviruses can cause bronchiolar and interstitial necrosis. A combination of measles and adenovirus produces the most severe symptoms, the most prolonged course and the highest mortality.
2. *Laryngotracheobronchitis* may occasionally be severe and present with signs of upper airways obstruction (e.g. stridor).
3. *Encephalitis* can occur just before, during or after the rash. It may be due to direct invasion of the brain by the virus or by an allergic response to the infection. It can prove fatal.
4. *Hepatitis:* Young adults generally react unfavourably to measles. A few adult pts. who have severe disease may develop hepatitis with frank jaundice.
5. *Hemorrhagic measles* is very rare. Encephalopathy, bleeding, confluent hemorrhagic rash and pneumonia are present, and the condition is usually fatal.

Management

Antibiotics for lung infection. Treatment of secondary brochopneumonia must include an anti-staphylococcal

antibiotic. Vitamin A 200,000 IU for 2 days to children with severe measles will prevent ocular complications and reduce respiratory infections and measles-related mortality. *Uncommon complications in the post-measles state:* Gangrene of tips of fingers and toes, cancrum oris, septicemia, pyogenic infections, reactivation of pulmonary tuberculosis. Possible abortion or prematurity if infection occurs during pregnancy.

Chickenpox

The disease generally affects children under 10 years of age. Recovery is usually uneventful but complications, some of them serious, do occur.

1. *Pneumonitis* has an insidious onset and is more common in adults, and very young children. Tachypnoea, cyanosis, cough and haemoptysis signify severe disease. CXR shows widespread nodular infiltrates.
2. *Neurological complications:* Acute meningitis or encephalitis may develop soon after onset of the rash, but more commonly, an allergic encephalopathy develops, Reye's syndrome, especially if aspirin has been given to the child, transverse myelitis and Guillain-Barre syndrome may also occur.
3. *Hemorrhagic chickenpox* is rare but potentially fatal form of the disease. Extensive bleeding may occur into unaffected skin as well as into the rash.
4. *Immunocompromised child,* patient on corticosteroids. The disease is more severe.

Management

(a) Antibiotics for secondary infection. (b) Antiviral drug Acyclovir 20 mg/kg (up to 800 mg) qds in severe disease

with complication like pneumonitis. (c) Secondary infection may be averted by daily antiseptic baths.

Whooping Cough

Whooping cough is a highly infectious disease of the respiratory tract. With the introduction of vaccination there has been a decline in the incidence of the disease, but in children not immunised it remains a major health hazard. The disease can be potentially fatal particularly in young infants because of various complications.

Complications

1. *CNS complications:* Cerebral, subarachnoid and subdural hemorrhages are rare. Convulsions, mainly a result of cerebral anoxia, may occur in young infants and prove fatal.
2. *Respiratory:* Prolonged apnoeic episodes may occur before or after paroxysms of cough. Bronchopneumonia is due to secondary bacterial infection, infective organisms include *Strep. pneumoniae*, *H. influenzae* type B and group A β-hemolytic streptococci.
3. *Pressure effects of paroxysmal cough:* Common are epistaxis, subconjunctival hemorrhage, umbilical hernia, prolapse of rectum. Rarely pneumothorax.

Management

1. Hospitalization for all children less than 1 year of age and older children who look ill. ICU for infants with severe symptoms.
2. Erythromycin 50 mg/kg/24 hours to eliminate organisms from nasopharynx.
3. O_2 for recurrent or sustained hypoxemia.
4. Periodic gentle suction to remove secretions.

5. Nutrition: Small frequent feeds especially after a paroxysm. Adequate hydration.

Mumps

The mumps virus has a predilection for causing non-purulent inflammation in salivary glands and the meninges. It usually affects children and young adults. Serious complications include encephalomyelitis, orchitis and pancreatitis.

Complications

Complications of mumps can occur in absence of parotid swelling.

1. *Neurological:* (a) Aseptic meningitis is common and mimics that of enteroviral infections, except that the fever in mumps is higher and the CSF pleocytosis greater. (b) Encephalitis or encephalomyelitis is uncommon compared with meningitis but carries a more severe prognosis.
2. *Orchitis:* One or both testes become tender and inflamed a few days after onset of parotitis. Fever often recurs with severe testicular pain.
3. *Abdominal pain:* Nausea and vomiting with acute abdominal pain may occur early in the attack and should suggest oophoritis in women or pancreatitis in either sex. As the serum amylase level is usually raised in acute mumps, the test does not help in ruling out pancreatitis.

Management

In an infant with history of fits, temperature should be lowered by tepid sponging and paracetamol. For orchitis oral prednisolone 40 mg/day often reduces testicular pain

and swelling. In mumps meningitis, bed rest is advised until fever and headache have subsided.

Diphtheria

Diagnostic Features of Severe Diphtheria

1. No previous immunization against diphtheria.
2. Relative lack of pharyngeal pain.
3. Severe toxemia without high fever.
4. Extension of membrane beyond tonsillar fossa.
5. Diffuse cervical swelling.
6. Palatal paralysis.

Severe Types of Diphtheria

Pharyngeal diphtheria: In severe cases-foetor oris (nonspecific), hemorrhage and marked oedema of tissues, resulting in the appearance of 'bull-neck' diphtheria. General symptoms are severe with waxy pallor, extreme lassitude and drowsiness progressing to stupor.

Laryngeal diphtheria: Pharyngeal infection can spread to the larynx, or less commonly, can start there and, with further spread, can involve the trachea and bronchi. The symptoms resemble those of croup with cough, stridor and respiratory distress.

Management

1. *DAT:* The therapeutic dose is based on duration of symptoms and severity of the disease and ranges from 40,000 U for pharyngeal disease, and 150,000 U in malignant cases. Measures to combat anaphylaxis must be taken.

2. *Antibiotics:* Erythromycin 500 mg qds for patients allergic to the drug, given for 14 days. For children 10–15 mg/kg.

Management of Severe Complications

Complication	Clinical features and management
Respiratory obstruction	Aspiration of membrane in laryngeal diphtheria. May necessitate intubation tracheostomy
Myocarditis	Faint heart sounds, tictac rhythm, ST-T changes. Complete bed rest
Circulatory failure	Acute toxemia in severe case. Signs of shock
Cardiac failure	Low output failure, rapid weak pulse
Cardiac arrhythmias	Heart block, A-V dissociation. Cardiac pacing. High mortality
Bulbar paralysis	Muscles of swallowing paralysed with risk of aspiration, which may require intubation or tracheostomy. Tracheostomy may be preferable to intubation if risk of pushing membrane further into trachea
Respiratory paralysis	Diaphragm and intercostal muscles paralysed, which may require intubation and artificial ventilation

Infectious Mononucleosis

The IM is an acute self-limiting illness caused by primary infection with EBV virus, lasting for 2–3 weeks unless complications which may need special care.

Clinical Features

Fever which may be as high as 40°C, widespread maculopapular rash in some, periorbital oedema. Tonsillar enlargement with erythema and purulent exudate, which may occasionally be sufficiently severe to cause pharyngeal obstruction. Generalised lymphadenopathy, particularly cervical is common.

Diagnosis

(a) WBC $10–20 \times 10^9/1$ with increase in mononuclear cells of which 20% are atypical lymphocytes. (b) Nonspecific heterophile antibodies (positive Paul-Bunnel and monospot tests). (c) Specific antibody responses.

Complications though uncommon can be serious.

1. *Autoimmune hemolytic anemia:* Occasionally profound thrombocytopenia with purpura and hemorrhages.
2. *Splenic rupture:* Abdominal pain is uncommon in IM, and splenic rupture should be considered and excluded.
3. *Neurological:* Rarely encephalitis presenting with predominant cerebellar features, Guillain-Barre syndrome, transverse myelitis.
4. *Myocarditis.*

Management

No specific antiviral treatment. Corticosteroids may be prescribed in management of hemolytic anemia, thrombocytopenia, neurological complications and myocarditis. If tonsillar enlargement causes airway obstruction prednisolone 60 mg/day for 5–6 days. Tracheostomy may be required very occasionally.

Acute Disseminated Miliary Tuberculosis

Acute hematogenous TB can occur mainly in immunocompromised individuals and victims of AIDS.

Clinical Features

Patient may present as pyrexia of unknown origin, or with tachypnoea, lymphadenopathy, tuberculoma of brain or multiple organ dysfunction, palpable liver and spleen, liver dysfunction with increased serum bilirubin, anemia and hypoproteinemia.

Investigation

(a) Repeated blood cultures for gram-negative infections. (b) Fibreoptic bronchoscopy (if pulmonary involvement) with bronchial aspirates for acid-fast bacilli. (c) Liver biopsy for presence of caseating granulomas. (d) CT scan of head for tuberculoma. (e) CXR may show miliary lung shadows.

Management

1. *Antituberculous therapy:* INH, RIF, PZI and EMB with addition of one or more second line drugs with a fluoroquinolone and an aminoglycoside or capromycin.
2. *Circulatory support:* (a) Fluid and electrolyte balance. (b) Dopamine. (c) Packed cell transfusion. (d) IV nutrition if GI intolerance, or via nasogastric tube if patient too weak to swallow.