

# Causal analysis and treatment protocols for vector-borne diseases

VECTOR CONTROL RESEARCH CENTRE, PONDICHERRY

## 1. Malaria

**Table 1.1** Causes, symptoms and treatment of malaria

Condition	Symptoms	Causes	Treatment
Falciparum malaria ('malignant' tertian or subtertian)	<ul style="list-style-type: none"> <li>• Febrile paroxysms</li> <li>• Intermittent chills rather than a clearly circumscribed cold phase, hot and sweating stages</li> <li>• Fever is remittent, daily or once in 3 days or twice every 3 days associated with chills</li> <li>• Non-specific symptoms such as fever, prostration, postural hypotension, a tinge of jaundice and tender hepatosplenomegaly</li> </ul>	Malarial parasite: <i>Plasmodium falciparum</i>	<ul style="list-style-type: none"> <li>• Treatment following microscopic confirmation</li> <li>• Chloroquine (CQ) 1500 mg orally in divided doses for 3 days in CQ-sensitive areas</li> <li>• Primaquine 45 mg single dose</li> <li>• In CQ-resistant areas—single dose of sulphadoxine (1500 mg) + pyrimethamine (7 mg) followed by primaquine (45 mg)</li> <li>• Appropriate management for other symptoms</li> </ul>
Cerebral malaria	<ul style="list-style-type: none"> <li>• History of several days of fever and non-specific symptoms as mentioned above</li> <li>• Complicated with gradual impairment of consciousness or generalized convulsions followed by persisting coma</li> <li>• Other manifestations (hyperpyrexia, hyperparasitaemia, hypoglycaemia, renal failure, hepatic dysfunction, pulmonary oedema, algid malaria, black water fever, etc.)</li> </ul>	Malarial parasite: <i>Plasmodium falciparum</i>	<ul style="list-style-type: none"> <li>• As in the case of falciparum malaria, treatment following microscopic confirmation</li> <li>• Supportive therapy (oxygen, and maintaining fluid and electrolyte balance)</li> <li>• Quinine IV drip (600 mg) repeated two or three times in 24 hours or</li> <li>• Artemether IV/IM (3.2 mg per kg on day 1; 1.6 mg per kg days 2–6)</li> <li>• CQ 1500 mg orally in divided doses for 3 days in CQ-sensitive areas</li> <li>• Primaquine 45 mg in single dose</li> <li>• In CQ-resistant areas: sulphadoxine–pyrimethamine compound—sulphadoxine (500 mg tablet) or sulphalene (500 mg) plus pyrimethamine (25 mg)—3 tablets as a single dose</li> </ul>
Vivax malaria ('benign' tertian malaria)	<ul style="list-style-type: none"> <li>• Intermittent fever with cold, hot and sweating stages; associated symptoms include headache, pain in the back, nausea and general malaise (the prodromal symptoms are mild or absent in relapses)</li> <li>• Febrile paroxysms occur in cycles of two days</li> </ul>	Malarial parasite: <i>Plasmodium vivax</i>	<ul style="list-style-type: none"> <li>• Treatment following microscopic confirmation</li> <li>• CQ 1500 mg orally in divided doses for 3 days</li> <li>• Primaquine 75 mg in 5 divided doses</li> </ul>
Malariae malaria ('quartan' malaria)	<ul style="list-style-type: none"> <li>• Clinical picture resembles that of vivax malaria but prodromal symptoms and rigors may be more severe</li> <li>• Anaemia is less pronounced</li> <li>• Febrile paroxysms occur in cycles of three days</li> </ul>	Malarial parasite: <i>Plasmodium malariae</i>	<ul style="list-style-type: none"> <li>• Treatment following microscopic confirmation</li> <li>• CQ 1500 mg orally in divided doses for 3 days</li> <li>• Primaquine 45 mg in a single dose</li> </ul>
Ovale malaria (ovale tertian malaria)	<ul style="list-style-type: none"> <li>• Clinical picture resembles that of vivax malaria but prodromal symptoms may be severe</li> <li>• Spontaneous recovery is more common with fewer relapses</li> <li>• Febrile paroxysms occur in cycles of two days</li> </ul>	Malarial parasite: <i>Plasmodium ovale</i>	<ul style="list-style-type: none"> <li>• Treatment following microscopic confirmation</li> <li>• CQ 1500 mg orally in divided doses for 3 days</li> <li>• Primaquine 75 mg in 5 divided doses</li> </ul>

**Table 1.2** Causes of conditions (by significance) due to malaria

Condition		Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)	
Falciparum malaria ('malignant' tertian or subtertian)	Main causes— <i>Plasmodium falciparum</i>	Multiplication of the malarial parasite and destruction of RBCs	Lack of nutrition, especially in children and pregnant women	<ul style="list-style-type: none"> <li>Poor economic conditions leading to creation of mosquito-genic condition favouring transmission</li> <li>Occupation, sleeping habits leading to increased man/vector contact</li> </ul>	<ul style="list-style-type: none"> <li>Low literacy associated with poor economic status leads to constraints in practising personal protection measures and seeking health care</li> </ul>
Cerebral malaria	<i>Plasmodium falciparum</i>	<ul style="list-style-type: none"> <li>Multiplication of the malarial parasite and destruction of RBCs</li> <li>Increased intravascular permeability/blockade of cerebral capillaries due to high parasitaemia leading to cerebral infarction, etc.</li> </ul>			Lack of adequate and timely treatment leading to cerebral malaria
Vivax malaria ('benign' tertian malaria)	<i>Plasmodium vivax</i>	Multiplication of the malarial parasite and destruction of RBCs			Relapses in vivax malaria
Malariae malaria ('quartan' malaria)	<i>Plasmodium malariae</i>	Multiplication of the malarial parasite and destruction of RBCs			
Ovale malaria (ovale tertian malaria)	<i>Plasmodium ovale</i>	Multiplication of the malarial parasite and destruction of RBCs			
	Interaction with other causes	Anaemia			

References: Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.  
Bruce-Chwatt LJ. *Essential malariology*. London: William Heinmann Medical Books Ltd; 2002:85–126.

**Table 1.3** Interventions (by significance) for various outcomes of malaria

Condition	Outcomes	Medical interventions	Non-medical interventions		
			Exercise	Nutrition	Others
Malarial fever (all the four plasmodium parasites)	Malaria and anaemia	<ul style="list-style-type: none"> <li>Antimalarials</li> <li>Antipyretics, analgesics</li> <li>Oral fluids and electrolytes</li> </ul>	Nil	Balanced nutritious diet, vitamin supplements	<ul style="list-style-type: none"> <li>Bed rest</li> <li>Sponging</li> </ul>
	Cerebral malaria	<ul style="list-style-type: none"> <li>Antimalarials</li> <li>Symptomatic</li> <li>Oxygen therapy</li> <li>Fluid and electrolyte balance</li> </ul>			<ul style="list-style-type: none"> <li>Bed rest</li> <li>Good nursing care</li> </ul>

**Table 1.4** Standard treatment protocols for malaria

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Malarial fever (paroxysms due to any one of the <i>Plasmodium</i> parasites)	<ul style="list-style-type: none"> <li>Trained nurses</li> <li>Medical practitioner (1 week)</li> </ul>	<ul style="list-style-type: none"> <li>Clinical signs and symptoms</li> <li>Peripheral blood examination</li> <li>ICT malaria test Pf serology</li> <li>Molecular biological techniques (DNA/RNA probes)</li> </ul>	<ul style="list-style-type: none"> <li>Chloroquine (CQ): In CQ-sensitive areas, 1500 mg orally in divided doses for 3 days</li> <li>Primaquine 45 mg in single dose for Pf and Pm, and 75 mg in 5 divided doses for Pv and Po</li> <li>SP compounds in other areas</li> </ul>	May/may not be required
Cerebral malaria	<ul style="list-style-type: none"> <li>Medical practitioner</li> <li>Referral hospitals (2 weeks)</li> </ul>	<ul style="list-style-type: none"> <li>Clinical signs and symptoms</li> <li>Peripheral blood examination</li> <li>ICT malaria test Pf serology</li> <li>Molecular biological techniques (DNA/RNA probes)</li> </ul>	<ul style="list-style-type: none"> <li>Antimalarials (quinine, artemisinin, CQ depending on the indications)</li> <li>Fluid replacement</li> <li>Oxygen therapy when needed</li> <li>Supportive therapy</li> </ul>	Mandatory

References: Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.  
Bruce-Chwatt LJ. *Essential malariology*. London: William Heinmann Medical Books Ltd; 2002:85–126.

Pf: *Plasmodium falciparum*; Pm: *Plasmodium malariae*; Pv: *Plasmodium vivax*; Po: *Plasmodium ovale*; SP: sulphadoxine–pyrimethamine; ICT: immunochromatographic card test

## 2. Dengue

**Table 2.1** Causal analysis of dengue

Condition	Symptoms	Causes	Treatment
Asymptomatic virus carriers	Asymptomatic	Dengue virus (serotypes 1–4)	No treatment available
Dengue fever (DF)	<ul style="list-style-type: none"> <li>Suspected: Compatible with clinical description (2 or more of the following clinical features—headache, retro-orbital pain, myalgia, arthralgia, rash, haemorrhagic manifestations)</li> <li>Probable: compatible with clinical description and either 1 or more of supportive serology (HI &gt;1280), anti-dengue IgG/IgM, same location/same time as confirmed cases</li> <li>Confirmed: Compatible with clinical description and laboratory tests confirmed</li> </ul>	Dengue virus (serotypes 1–4)	<ul style="list-style-type: none"> <li>Symptomatic (antipyretics, analgesics)</li> <li>Bed rest</li> <li>Sponging</li> <li>Oral fluids and electrolytes</li> </ul>
Dengue haemorrhagic fever (DHF)	Probable and confirmed: one or more of positive tourniquet test, petechiae, ecchymosis or purpura, bleeding from mucosa, gastrointestinal tract, injection sites, haematemesis or melaena and thrombocytopenia (platelets <100,000/cmm), and evidence of plasma leakage (>20% rise in haematocrit, pleural effusion/ascites)	Dengue virus (serotypes 1–4)	<ul style="list-style-type: none"> <li>Symptomatic</li> <li>Fluid replacement</li> <li>Whole blood/platelet replacement</li> </ul>
Dengue shock syndrome (DSS)	Same as DHF and evidence of circulatory failure manifested as rapid and weak pulse, pulse pressure <20 mmHg, etc.	Dengue virus (serotypes 1–4)	<ul style="list-style-type: none"> <li>Symptomatic</li> <li>Sedation</li> <li>Oxygen therapy</li> <li>Fluid/whole blood/platelet replacement</li> </ul>

**Table 2.2** Causes of conditions (by significance) due to dengue

Condition		Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Dengue fever	Main causes	<ul style="list-style-type: none"> <li>• Non-specific 'viral syndrome' with fever, headache, myalgia, chills, arthralgia, retro-orbital pain with or without rash</li> <li>• Virus multiplication in macrophages</li> </ul>	Nil	<ul style="list-style-type: none"> <li>• Practice of storing water in containers for domestic purposes</li> <li>• Low literacy associated with poor economic status leads to constraints in practising personal protection measures</li> </ul>
	Interaction with other causes	Nil		
Dengue haemorrhagic fever	Main causes	<ul style="list-style-type: none"> <li>• One or more of positive tourniquet test, petechiae, ecchymosis or purpura, bleeding from mucosa, gastro-intestinal tract, injection sites, haematemesis or melaena and thrombocytopenia (platelets &lt;100,000/cmm), and evidence of plasma leakage (&gt;20% rise in haematocrit, pleural effusion/ascites)</li> <li>• Increased vascular permeability resulting in plasma leakage (mainly into the peritoneal and pleural cavities), hypovolaemia and shock</li> <li>• Abnormal haemostasis due to vasculopathy, thrombocytopenia</li> <li>• Complement C3 and C5 levels depressed, C3a and C5a levels elevated</li> </ul>	Nil	<ul style="list-style-type: none"> <li>• Practice of storing water in containers for domestic purposes</li> <li>• Low literacy associated with poor economic status leads to constraints measures, partial immunity, in practising personal protection monsoon and post-monsoon (of late, dry summer season also)</li> <li>• Light to moderate rainfall, plains and plateau</li> <li>• Temperature: 20–25 °C/relative humidity &gt;60%, both urban poor and rural areas favourable; urban: due to poor management of immediate environment, rural: poor water storage practices at household level in areas with erratic water supply contribute to breeding source for the incriminated vectors</li> </ul>
	Interaction with other causes	Nil		
Dengue shock syndrome	Main causes	<ul style="list-style-type: none"> <li>• Same as DHF and evidence of circulatory failure manifested as rapid and weak pulse, pulse pressure &lt;20 mmHg, etc.</li> <li>• Increased vascular permeability resulting in plasma leakage (mainly into the peritoneal and pleural cavities), hypovolaemia and shock</li> <li>• Abnormal haemostasis due to vasculopathy, thrombocytopenia</li> <li>• Complement C3 and C5 levels depressed, C3a and C5a levels elevated</li> </ul>	Nil	<ul style="list-style-type: none"> <li>• Practice of storing water in containers for domestic purposes</li> <li>• Same as for DHF</li> </ul>
	Interaction with other causes	Nil		

References: World Health Organization. *Prevention and control of dengue and dengue haemorrhagic fever: Comprehensive guidelines*. WHO regional publication, SEARO; 1999:3–53.

Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

**Table 2.3** Interventions for various outcomes of dengue (by significance)

Outcome of dengue	Medical interventions	Non-medical interventions		
		Exercise	Nutrition	Others
Dengue fever	<ul style="list-style-type: none"> <li>• Symptomatic (antipyretics, analgesics)</li> <li>• Oral fluid and electrolytes</li> </ul>	Nil	Nil	<ul style="list-style-type: none"> <li>• Bed rest</li> <li>• Sponging</li> </ul>
Dengue haemorrhagic fever	<ul style="list-style-type: none"> <li>• Symptomatic</li> <li>• Fluid replacement</li> <li>• Whole blood/platelet replacement</li> </ul>			Good nursing care
Dengue shock syndrome	<ul style="list-style-type: none"> <li>• Symptomatic</li> <li>• Sedation</li> <li>• Oxygen therapy</li> <li>• Fluid/whole blood/platelet replacement</li> </ul>			<ul style="list-style-type: none"> <li>• Bed rest</li> <li>• Good nursing care</li> </ul>

**Table 2.4** Standard treatment protocols for dengue

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Dengue fever (DF)	<ul style="list-style-type: none"> <li>• Trained nurses</li> <li>• Medical practitioner</li> </ul>	<ul style="list-style-type: none"> <li>• Haemagglutination inhibition (HI) test</li> <li>• Complement fixation test</li> <li>• Neutralization test (NT)</li> <li>• ELISA for IgG and IgM</li> <li>• Isolation of the virus</li> </ul>	<ul style="list-style-type: none"> <li>• Antipyretics: 3–5 days</li> <li>• Analgesics: 3–5 days</li> </ul>	May not be required
Dengue haemorrhagic fever (DHF)	<ul style="list-style-type: none"> <li>• Medical practitioner</li> <li>• Hospital</li> </ul>	<ul style="list-style-type: none"> <li>• Clinical signs and symptoms</li> <li>• ELISA for IgG and IgM</li> <li>• Isolation of the virus</li> </ul>	<ul style="list-style-type: none"> <li>• Analgesics</li> <li>• Antipyretics</li> <li>• Fluid replacement</li> <li>• Whole blood/platelet replacement</li> </ul>	Must
Dengue shock syndrome (DSS)	<ul style="list-style-type: none"> <li>• Medical practitioner</li> <li>• Referral hospital</li> </ul>	<ul style="list-style-type: none"> <li>• Clinical signs and symptoms</li> <li>• ELISA for IgG and IgM</li> <li>• Isolation of the virus</li> </ul>	<ul style="list-style-type: none"> <li>• Analgesics</li> <li>• Antipyretics</li> <li>• Fluid replacement</li> <li>• Whole blood/platelet/plasma/replacement</li> </ul>	Must

References: World Health Organization. *Prevention and control of dengue and dengue haemorrhagic fever: Comprehensive guidelines*. WHO regional publication, SEARO; 1999:3–53.

Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

### 3. Japanese encephalitis

**Table 3.1** Causal analysis of Japanese encephalitis

Condition	Symptoms	Causes	Treatment
Prodromal	<ul style="list-style-type: none"> <li>• Fever</li> <li>• Rigors</li> <li>• Headache</li> <li>• Nausea</li> <li>• Vomiting</li> </ul>	JE virus (Flaviviridae family)	<ul style="list-style-type: none"> <li>• Non-specific treatment, only symptomatic and supportive treatment is crucial</li> <li>• Should NOT use gamma-globulins and corticosteroids; physical therapy desirable</li> </ul>
Acute encephalitic	<ul style="list-style-type: none"> <li>• Convulsions</li> <li>• Neck rigidity</li> <li>• Muscular rigidity</li> <li>• Mask-like face</li> <li>• Abnormal movements</li> <li>• Dehydration</li> </ul>	JE virus (Flaviviridae family)	(Supportive) <ul style="list-style-type: none"> <li>• 1/2 normal saline infusion (children)</li> <li>• IV diazepam (0.3 mg/kg, every 4–6 hours) or phenobarbital (10%, 0.5 to 1 ml IM)</li> <li>• Antipyretic suppositories used</li> <li>• Cold/tepid sponging</li> <li>• Oxygen (1–2 litres/minute), if needed</li> </ul>
Late stage	<ul style="list-style-type: none"> <li>• Increased deep tendon reflexes</li> <li>• Thick and slow speech</li> <li>• Aphasia and paresis</li> </ul>	JE virus (Flaviviridae family)	Rehabilitation during convalescence with physiotherapy

**Table 3.2** Causes of conditions (by significance) due to Japanese encephalitis

Causes	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)	
Main causes	<ul style="list-style-type: none"> <li>• Viral multiplication in the local and regional lymph nodes</li> <li>• Dissemination to secondary sites and further multiplication leading to viraemia and invasion of the central nervous system</li> </ul>	Nil	<ul style="list-style-type: none"> <li>• Poor intersectoral coordination between health, agriculture, animal husbandry and local administration departments</li> <li>• Intensified wet agriculture activities facilitating breeding of vectors in rice fields, and pig (amplifier hosts) rearing favour transmission</li> <li>• Poor community participation in primary health care with respect to vector-borne disease control activities</li> </ul>	<ul style="list-style-type: none"> <li>• Illiteracy and ignorance about JE transmission leading to constraints in practising personal protection measures</li> <li>• <i>Culex</i> group of mosquitoes coming in contact with virus-infected birds</li> </ul>
Interaction with other causes	No recorded reports			

Reference: Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

**Table 3.3** Interventions (by significance) for Japanese encephalitis

Outcome	Medical interventions	Non-medical interventions		
		Exercise	Nutrition	Other
Prodromal	Supportive <ul style="list-style-type: none"> <li>• IV fluids</li> <li>• Antipyretics</li> </ul>	If needed	Yes	—
Encephalitic	Supportive <ul style="list-style-type: none"> <li>• IV fluids</li> <li>• Antipyretics</li> <li>• Anticonvulsants</li> <li>• Tepid sponging</li> </ul>	Yes	Yes	—

Reference: Rao PN. *Japanese encephalitis*. 2002.

**Table 3.4** Standard treatment protocols for Japanese encephalitis

Condition	Personnel	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay	Non-medical interventions/prevention
Prodromal	Medical practitioner and nurse	<ul style="list-style-type: none"> <li>• Serum and CSF</li> <li>• IgM—capture ELISA (MAC ELISA)</li> <li>• Immunofluorescent assay (IFA)</li> </ul>	Supportive <ul style="list-style-type: none"> <li>• IV fluids</li> <li>• Antipyretics</li> <li>• Anticonvulsants</li> <li>• Tepid sponging</li> </ul>	Until complete recovery	Rest and good nutrition during convalescence, physiotherapy for neurological residual sequelae, if any, after complete recovery
Encephalitic	Medical practitioner	Reverse passive haemagglutination for confirmation			

(Cont.)

**Table 3.4** (cont.) Standard treatment protocols for Japanese encephalitis

Condition	Personnel	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay	Non-medical interventions/prevention
		<ul style="list-style-type: none"> <li>• Detection of the JE virus, antigen or genome in the tissue, blood or other body fluid by immunochemistry or immunofluorescence or PCR, or</li> <li>• JE virus-specific IgM in the CSF, or</li> <li>• Four-fold or greater rise in JE virus-specific antibody in paired sera (acute and convalescent phase) through IgM/IgG, ELISA, haemagglutination inhibition test, in a patient with no history of recent yellow fever vaccination and where cross-reactions to other flaviviruses have been excluded</li> </ul>			

Reference: Rao PN. *Japanese encephalitis*. 2002.

### 1. General features

- Zoonotic viral disease
- Maintained in nature in animals and birds (pigs, egrets, pond herons)
- Transmitted primarily by mosquitoes of the genus *Culex*
- Human infection is only incidental, mostly not apparent, with 1 in 250–1000 infections resulting in disease
- Case fatality rate 20% and above; can be kept low by good management
- Until the early 1970s, reported only from south India; by 1978, from 21 States and UTs. Worst outbreak in 1988 in UP
- 10 states report incidence of JE regularly (AP, UP, Karnataka, TN, WB, Assam, Bihar, Goa, Manipur and Haryana)
- Population at risk >160 million
- Seasonal (May–October)
- Related to agricultural practices
- Children usually affected, adults affected in outbreaks in non-endemic areas
- Males affected more than females
- In endemic areas, 12%–44% of pigs are positive for the JE virus

### 2. Clinical features

There are three stages of JE:

- **Prodromal stage:** This stage is characterized by fever, rigors, headache, and nausea and vomiting. The stage lasts for 1–6 days.

- **Acute encephalitic stage:** This stage begins by the 3rd–5th day and is characterized by convulsions, altered sensorium, mask-like face, stiff neck, muscular rigidity, tremors (fingers, tongue, eyelids, eyes), abnormal movements of the limbs and speech impairment
- **Late stage:** During this stage, there is persistence of central nervous system (CNS) injury signs such as mental impairment, increased deep tendon reflexes, paresis, speech impairment, epilepsy, abnormal movements and behaviour abnormalities

### 3. Diagnosis of JE

- Collect the cerebrospinal fluid (CSF) (by lumbar puncture, in early acute phase, store at 4 °C in dry ice, transport to laboratory)
- Collect brain tissue from dead patients (immerse in 2 ml of 10% glycerol–saline (pH 7.4), transport in a sealed container in dry ice or liquid nitrogen for isolation of the virus)
- Collect blood (in acute phase and convalescence, serum to be separated and stored at 4 °C)

### 4. Case definition

Infection caused by JE virus may result in a febrile illness of variable severity associated with neurological symptoms ranging from headache to meningitis or encephalitis. Symptoms include headache, fever, meningeal signs, stupor, disorientation, coma, tremors, paresis (generalized),

**Table 3.5** Year-wise and State-wise distribution of cases of Japanese encephalitis

State	1992		1993		1994		1995		1996		1997		1998		1999		2000		2001	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
Andhra Pradesh	143	66	1175	467	260	131	986	270	332	108	982	247	527	192	965	200	343	72	0	0
Assam	259	93	96	30	230	145	11	11	64	29	88	26	26	6	11	2	158	69	241	113
Bihar	0	0	0	0	0	0	0	0	0	—	—	—	—	—	—	—	77	19	88	16
Delhi	352	103	106	49	54	31	3	1	—	—	1	—	—	—	—	—	—	—	1	0
Goa	43	8	37	10	10	1	11	3	4	0	40	8	0	0	0	0	15	3	2	0
Gujarat	0	0	0	0	0	0	0	0	—	—	1	—	—	—	—	—	—	—	0	0
Haryana	41	33	19	13	79	67	30	21	59	41	0	0	19	16	121	56	74	43	45	22
Karnataka	58	15	99	22	126	47	285	89	171	17	436	87	306	50	597	88	438	45	73	6
Kerala	0	0	0	0	0	0	0	0	105	31	121	19	103	24	214	4	164	2	0	0
Maharashtra	0	0	0	0	0	0	0	0	—	—	43	3	2	0	NA	NA	—	—	0	0
Manipur	29	0	11	1	11	0	268	62	20	2	0	0	28	1	42	1	1	0	0	0
Tamil Nadu	177	107	278	176	239	125	115	57	111	53	89	42	25	14	14	5	4	0	0	0
Uttar Pradesh	793	229	104	32	0	0	0	0	672	161	351	76	1051	195	1370	275	1170	253	625	132
West Bengal	537	234	366	123	234	93	1265	428	706	151	362	124	36	9	61	14	148	50	97	14
Total	2432	888	2291	923	1243	640	2974	942	2244	593	2514	632	2123	507	3395	645	2592	556	1172	303
CFR%	36.51		40.29		51.49		31.67		26.43		25.14		23.88		19.00		21.45		25.85	

NA: not applicable

Source: National Institute of Communicable Diseases, Delhi

hypertonia, loss of coordination. Clinically, the encephalitis cannot be distinguished from other CNS infections.

### 5. Detection and isolation of the virus

#### Detection of antigen from CSF

- Immunofluorescent assay (IFA) (can detect antigen as early as second day; useful when IgM antibodies are not detected in CSF)
- Reverse passive haemagglutination (RPHA) (within 10 days of onset)
- PCR (for virus genome)
- Inoculation into infant mice

#### Detection of antibody

From serum

- Haemagglutination inhibition (HI)
- Complement fixation (CF), kinetic CF
- Neutralization test
- Immunodiffusion

All based on a rise of IgG antibody titre (four-fold) in paired sera collected at 15–20 days' interval

#### Detection of antibody from serum and CSF

- IgM captured enzyme-linked immunosorbent assay (MAC ELISA) (IgM antibodies present after 7th day of illness)
- A single specimen is sufficient

### 6. Management of JE (only symptomatic)

- Fluid–electrolyte balance

- Temperature
- Convulsions
- Intracranial pressure

### 7. JE control strategy

- Surveillance
  - sero surveillance in animals/birds
  - vector surveillance (vector density, infection)
  - case surveillance
- 1. Where no JE transmission is detected but vector is present (acute CNS syndromes, fever clustering)
- 2. Where endemic or epidemic (weekly/monthly reporting on suspected/probable/confirmed cases)
- Vaccination
- Vector control (indoor residual spraying, outdoor fogging)
- Diagnosis and management (including rehabilitation)
- Health education/distribution of information, education and communication (IEC) materials
- Training (health sector, other sectors)

### 8. JE vaccine

Three types of vaccines for JE are produced and used worldwide:

- Inactivated mouse brain vaccine (India–Nakayama strain) (0.5–1 ml SC, 2 doses, 1–4 weeks apart, booster after 1 year, subsequent boosters at 1–3-year intervals)
- Inactivated hamster kidney cell vaccine
- Live-attenuated hamster kidney cell vaccine

## 4. Kala-azar

**Table 4.1** Causes, symptoms and treatment of kala-azar

Condition	Symptoms	Cause	Treatment
Kala-azar (visceral leishmaniasis)	<ul style="list-style-type: none"> <li>• Fever</li> <li>• Hepatosplenomegaly</li> <li>• Anaemia</li> <li>• Wasting</li> <li>• Pigmentation</li> <li>• Cough</li> <li>• Diarrhoea</li> </ul>	<i>Leishmania donovani</i>	<ul style="list-style-type: none"> <li>• Antimony compounds 10 mg/kg IV/IM for 20 days</li> <li>• Antimony compounds 20 mg/kg IV/IM for 20 days</li> <li>• Liposomal amphotericin B 2–3 mg/kg/dose/day for 10 days</li> </ul>
Post kala-azar dermal leishmaniasis (PKDL)	<ul style="list-style-type: none"> <li>• Hypopigmented erythematous macules</li> <li>• Nodular eruptions on the face</li> </ul>	<i>Leishmania donovani</i>	Same as above
Cutaneous leishmaniasis	Nodule at the site of inoculation, central crust development, gradual healing	<i>Leishmania tropica</i>	Same as above

IV: intravenous; IM: intramuscular

**Table 4.2** Causes of conditions (by significance) due to kala-azar

Condition		Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Kala-azar (VL)	Main causes	<ul style="list-style-type: none"> <li>• Multiplication of <i>Leishmania</i> in macrophages of liver, spleen, bone marrow and lymphoid tissue</li> <li>• Granulopenia and thrombocytopenia</li> </ul>	Nil	<ul style="list-style-type: none"> <li>• Poor economic conditions leading to creation of vector breeding (sandfly) conditions favouring transmission</li> <li>• Low literacy associated with poor economic status leads to constraints in practising personal protection measures</li> </ul>
	Interaction with other causes		<ul style="list-style-type: none"> <li>• Anaemia</li> <li>• Immunosuppression</li> <li>• Association with other diseases such as TB and pneumococcal infection</li> </ul>	<ul style="list-style-type: none"> <li>• Mixed dwellings and proximity of human dwellings to cattle sheds</li> </ul>
PKDL	Main causes	<ul style="list-style-type: none"> <li>• Pathology same as above</li> <li>• Usually one to several years after apparent recovery from VL</li> <li>• Hypopigmented erythematous macules</li> <li>• Nodular eruptions on the face</li> </ul>		<ul style="list-style-type: none"> <li>• Poor economic conditions leading to creation of mosquito-genic conditions favouring transmission</li> <li>• Low literacy associated with poor economic status leads to constraints in practising personal protection measures</li> </ul>
	Interaction with other causes		<ul style="list-style-type: none"> <li>• Anaemia</li> <li>• Immunosuppression</li> <li>• Association with other diseases such as TB and pneumococcal infection</li> </ul>	
Cutaneous leishmaniasis (CL)	Main causes	<ul style="list-style-type: none"> <li>• Host immunity—immune cellular response</li> <li>• Tissue response (tissue damage due to antigen release)</li> <li>• Nodule at the site of inoculation, central crust development, gradual healing</li> </ul>	Same as for PKDL	Same as for PKDL

References: Haslett C, Chilvers ER, Boon NA, Colledge NR (eds). *Davidson's principles and practice of medicine*. 19th ed. Churchill Livingstone; 2002:66–8.

World Health Organization. *Technical Report Series-701: Report of a WHO Expert Committee*. Geneva: WHO; 1984:14–23.

*Manson's tropical diseases*. 17th ed. USA: Williams and William; 1976:117.

Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

PKDL: post kala-azar dermal leishmaniasis; VL: visceral leishmaniasis

**Table 4.3** Interventions for various outcomes of kala-azar (by significance)

Outcomes	Medical interventions	Non-medical interventions		
		Exercise	Nutrition	Others
VL	<ul style="list-style-type: none"> <li>• Antimony compounds</li> <li>• Antipyretics</li> <li>• Amphotericin B</li> </ul>	Nil	Correction of anaemia	Recurrent pneumococcal infection
PKDL	<ul style="list-style-type: none"> <li>• Antimony compounds</li> <li>• Antipyretics</li> <li>• Amphotericin B</li> </ul>		Correction of anaemia	
Cutaneous leishmaniasis	<ul style="list-style-type: none"> <li>• Antimony compounds</li> <li>• Antipyretics</li> <li>• Amphotericin B</li> </ul>		Correction of anaemia	

References: Haslett C, Chilvers ER, Boon NA, Colledge NR (eds). *Davidson's principles and practice of medicine*. 19th ed. Churchill Livingstone; 2002:66–8.

World Health Organization. *Technical Report Series-701: Report of a WHO Expert Committee*. Geneva: WHO; 1984:14–23.

*Manson's tropical diseases*. 17th ed. USA: Williams and William; 1976:117.

Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

VL: visceral leishmaniasis; PKDL: post kala-azar dermal leishmaniasis

**Table 4.4** Standard treatment protocols for kala-azar

Condition	Personnel	Tests (by type)	Drugs (dosage,type and time)	Inpatient stay	Others
Kala-azar	Medical practitioner	<ul style="list-style-type: none"> <li>LD bodies</li> <li>Aldehyde tests</li> <li>Serological tests</li> <li>Leishmanin test</li> <li>Haematological tests (leucopenia, anaemia, reversed albumin–globulin ratio,increased ESR, RBC–WBC ratio)</li> </ul>	<ul style="list-style-type: none"> <li>Antimony compounds 10 mg/kg IV/IM for 20 days</li> <li>Antimony compounds 20 mg/kg IV/IM for 20 days</li> <li>Liposomal amphotericin B 2–3 mg/kg/dose/day for 10 days</li> </ul>	Required initially	Nil
PKDL	Medical practitioner	As above	As above	Not required	
Cutaneous leishmaniasis	Medical practitioner	As above	As above	Not required	

References: Haslett C, Chilvers ER, Boon NA, Colledge NR (eds). *Davidson's principles and practice of medicine*. 19th ed. Churchill Livingstone; 2002:66–8. World Health Organization. *Technical Report Series-701: Report of a WHO Expert Committee*. Geneva: WHO; 1984:14–23. *Manson's tropical diseases*. 17th ed. USA: The Williams and William; 1976:117. Park K (ed). *Park's textbook of preventive medicine*. Jabalpur: M/S Banarasidas Bhanot; 2000:172–3, 215.

LD: Leishman–Donovan; IV: intravenous; IM: intramuscular; ESR: erythrocyte sedimentation rate; RBC: red blood cell; WBC: white blood cell; PKDL: post kala-azar dermal leishmaniasis

## 5. Lymphatic filariasis

**Table 5.1** Symptoms, causes and treatment of lymphatic filariasis

Condition	Symptoms	Causes	Treatment
Mf carriers	Asymptomatic	Circulating microfilaria in the peripheral blood	One course of diethylcarbamazine (DEC) at 6 mg/kg body weight per day for 12 days in three daily doses
ADL	Fever associated with adenolymphangitis	Secondary bacterial infection at the sites damaged by the filarial parasite in the lymphatic system	<ul style="list-style-type: none"> <li>Antibiotics (choice penicillin)</li> <li>Antipyretics</li> <li>Anti-inflammatory drugs</li> </ul>
TPE	<ul style="list-style-type: none"> <li>Dry nocturnal cough</li> <li>Exertional dyspnoea</li> <li>Absolute eosinophil count more than 3000/cmm</li> <li>Differential count more than 25%</li> </ul>	Immunological (hyper) response	<ul style="list-style-type: none"> <li>Anthelmintic drug</li> <li>One course of DEC at 6 mg/kg body weight per day for 12 days in three daily doses</li> <li>Non-responders?</li> </ul>
Lymphoedema I	<ul style="list-style-type: none"> <li>Reversible oedema in the limbs overnight</li> <li>Skin normal</li> </ul>	Damage to the lymphatic system (filarial parasite)	<ul style="list-style-type: none"> <li>Limb elevation</li> <li>Manual massage</li> <li>DEC?</li> </ul>
Lymphoedema II	<ul style="list-style-type: none"> <li>Irreversible oedema in the limbs even with elevation</li> <li>Skin normal</li> </ul>	Repeated attacks of ADL due to filarial infection	<ul style="list-style-type: none"> <li>Limb hygiene, limb elevation, management of ADL</li> <li>Physiotherapy</li> </ul>
Lymphoedema III	<ul style="list-style-type: none"> <li>Irreversible oedema in the limbs</li> <li>Skin thickened</li> </ul>	Repeated attacks of ADL due to filarial infection	<ul style="list-style-type: none"> <li>Limb hygiene, elevation, management of ADL</li> <li>Physiotherapy</li> <li>Interferential therapy</li> </ul>
Lymphoedema IV	<ul style="list-style-type: none"> <li>Irreversible oedema in the limbs</li> <li>Skin-fold thickening, pigmentary changes, chronic ulceration and epidermal and subepidermal nodules</li> <li>Myiasis</li> </ul>	Repeated attacks of ADL due to filarial infection	<ul style="list-style-type: none"> <li>Topical application of antibiotics</li> <li>Interferential therapy</li> <li>Limb hygiene, elevation, management of ADL</li> <li>Physiotherapy</li> <li>Lymphonodal shunt</li> </ul>
Hydrocele (filaricele) <15 cm	Swelling of the scrotum (tunica)	Accumulation of clear fluid in TVT due to filarial infection	Surgery
Hydrocele (filaricele) ≥15 cm	Swelling of the scrotum (tunica)	Accumulation of clear fluid in TVT due to filarial infection	Surgery
Chyluria	Milky urine	Obstruction to the lymphatic system	Antibiotics to prevent secondary infection

ADL: adenolymphangitis; TPE: tropical pulmonary eosinophilia; TVT: tunica vaginalis testis

**Table 5.2** Causes of conditions (by significance) due to lymphatic filariasis

Condition	Causes	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Lymphoedema I <sup>1-6</sup>	Main causes	<ul style="list-style-type: none"> <li>• Damage to the lymphatic system caused by the filarial parasite</li> <li>• Consequence of acute episodes of ADL</li> <li>• Accumulation of lymph in the lower extremities due to the defective lymphatic vessels</li> <li>• Oedema normally disappears on elevation</li> </ul>	<ul style="list-style-type: none"> <li>• Physical exertion</li> <li>• Occupational—weavers, tailors who tend to sit for a long time with lower limbs in more exertion</li> </ul>	<ul style="list-style-type: none"> <li>• Poor economic conditions leading to creation of mosquitogenic conditions favouring transmission</li> <li>• Low literacy associated with poor economic status leads to constraints in practising personal protection measures</li> </ul>
	Interaction with other causes	Nil		
Lymph-oedema II <sup>1-4, 6, 7</sup>	Main causes	<ul style="list-style-type: none"> <li>• Damage to the lymphatic system caused by the filarial parasite</li> <li>• Obstructive and immunological effects</li> <li>• Pitting—oedema</li> <li>• Accumulation of lymph in the lower extremities due to defective lymphatic vessels</li> <li>• Oedema volume increases following each attack of ADL</li> </ul>	<ul style="list-style-type: none"> <li>• Physical exertion</li> <li>• Occupational—weavers, tailors who tend to sit for a long time with lower limbs in more exertion</li> </ul>	<ul style="list-style-type: none"> <li>• Poor economic conditions leading to creation of mosquitogenic conditions favouring transmission</li> <li>• Low literacy associated with poor economic status leads to constraints in practising personal protection measures</li> </ul>
	Interaction with other causes	Nil		
Lymph-oedema III <sup>1-4, 6-9</sup>	Main causes	<ul style="list-style-type: none"> <li>• Damage to the lymphatic system caused by the filarial parasite</li> <li>• Obstructive and immunological effects</li> <li>• Oedema—non-pitting</li> <li>• Loss of barrier facilitates penetration of bacteria</li> <li>• Accumulation of lymph in the lower extremities due to defective lymphatic vessels</li> <li>• Oedema volume increases following each attack of ADL</li> </ul>	<ul style="list-style-type: none"> <li>• Physical exertion</li> <li>• Occupational—weavers, tailors who tend to sit for a long time with lower limbs in more exertion</li> </ul>	<ul style="list-style-type: none"> <li>• Poor economic conditions leading to creation of mosquitogenic conditions favouring transmission</li> <li>• Low literacy associated with poor economic status leads to constraints in practising personal protection measures</li> </ul>
	Interaction with other causes	Bacteria enter the tissues through entry lesions in the skin		
Lymph-oedema IV <sup>1-4, 6-9</sup>	Main causes	<ul style="list-style-type: none"> <li>• Damage to the lymphatic system caused by the filarial parasite</li> <li>• Obstructive and immunological effects</li> <li>• Oedema—non-pitting</li> <li>• Loss of barrier function facilitates penetration of bacteria</li> <li>• Accumulation of lymph in the lower extremities due to defective lymphatic vessels</li> <li>• Skin-fold thickening</li> <li>• Hyperkeratosis</li> <li>• Hypo- or hypertrichosis</li> <li>• Pachyderma</li> <li>• Pigmentary changes</li> <li>• Chronic ulceration</li> <li>• Epidermal and subepidermal nodules</li> </ul>	<ul style="list-style-type: none"> <li>• Physical exertion</li> <li>• Occupational—weavers, tailors who tend to sit for a long time with lower limbs in more exertion</li> </ul>	<ul style="list-style-type: none"> <li>• Poor economic conditions leading to creation of mosquitogenic conditions favouring transmission</li> <li>• Low literacy associated with poor economic status leads to constraints in practising personal protection measures</li> </ul>

(Cont.)

**Table 5.2** (cont.) Causes of conditions (by significance) due to lymphatic filariasis

Condition	Causes	Direct (normally clinical)	Indirect (diet, exercise, alcohol)	Distant (sociopolitical, economic, empowerment, gender, literacy, etc.)
Hydrocele <sup>1-4,6-9</sup>	Interaction with other causes	Bacteria enter tissues through entry lesions in the skin		
	Main causes	<ul style="list-style-type: none"> <li>• Damage to the lymphatic system caused by the filarial parasite</li> <li>• Obstructive and immunological effects</li> <li>• Accumulation of lymph in the scrotal sac due to the defective lymphatic vessels</li> <li>• Skin-fold thickening</li> </ul>	<ul style="list-style-type: none"> <li>• Physical exertion</li> <li>• Occupational—vendors using bicycles</li> </ul>	<ul style="list-style-type: none"> <li>• Poor economic conditions leading to creation of mosquito-genic conditions favouring transmission</li> <li>• Low literacy associated with poor economic status leads to constraints in practising personal protection measures</li> </ul>
	Interaction with other causes	Bacteria enter tissues through entry lesions in the skin		

**Table 5.3** Interventions (by significance) for lymphatic filariasis

Outcomes	Medical interventions	Non-medical interventions		
		Exercise	Nutrition	Other
ADL	<ul style="list-style-type: none"> <li>• Antibiotics (choice penicillin)</li> <li>• Antipyretics</li> <li>• Anti-inflammatory drugs</li> </ul>			
TPE	<ul style="list-style-type: none"> <li>• Anthelmintic drugs</li> <li>• One course of DEC at 6 mg/kg body weight per day for 12 days in three daily doses</li> <li>• Non-responders?</li> </ul>			
Lymphoedema I	<ul style="list-style-type: none"> <li>• Elevation</li> <li>• Manual massage</li> <li>• DEC?</li> </ul>			
Lymphoedema II	<ul style="list-style-type: none"> <li>• Limb hygiene, elevation, management of ADL</li> <li>• Physiotherapy</li> </ul>	<ul style="list-style-type: none"> <li>• Limb elevation</li> <li>• Manual massage</li> </ul>		
Lymphoedema III	<ul style="list-style-type: none"> <li>• Limb hygiene, elevation, management of ADL</li> <li>• Physiotherapy</li> <li>• Interferential therapy</li> </ul>	<ul style="list-style-type: none"> <li>• Limb elevation</li> <li>• Manual massage</li> </ul>		
Lymphoedema IV	<ul style="list-style-type: none"> <li>• Topical application of antibiotics</li> <li>• Interferential therapy</li> <li>• Limb hygiene, elevation, management of ADL</li> <li>• Lymphonodal shunt</li> </ul>			
Hydrocele (filaricele) <15 cm	Surgery			
Hydrocele (filaricele) ≥15 cm	Surgery			
Chyluria	Antibiotics to prevent secondary infection			

ADL: adenolymphangitis; DEC: diethylcarbamazine; TPE: tropical pulmonary eosinophilia

**Table 5.4** Standard treatment protocols for lymphatic filariasis

Condition	Personnel (units of time and type)	Tests (by type)	Drugs (dosage, type and time)	Inpatient stay
Asymptomatic mf carriers <sup>1-4</sup>	Medical practitioner	Night blood (peripheral) examination	<ul style="list-style-type: none"> <li>One course of diethylcarbamazine at 6 mg/kg body weight per day for 12 days in three daily doses</li> </ul>	Not required
ADL <sup>1-4</sup>	Medical practitioner	Clinical signs and symptoms	<ul style="list-style-type: none"> <li>Antibiotics (choice penicillin)</li> <li>Antipyretics</li> </ul>	<ul style="list-style-type: none"> <li>Not required</li> <li>Anti-inflammatory drugs</li> </ul>
TPE <sup>1,10,11</sup>	Medical practitioner	Differential and absolute eosinophil count	<ul style="list-style-type: none"> <li>Anthelmintic drugs</li> <li>One course of DEC at 6 mg/kg body weight per day for 12 days in three daily doses</li> <li>Non-responders?</li> </ul>	Required in case of recurrent attacks and breathing difficulty
Lymphoedema I <sup>1-4</sup>	Medical practitioner	Clinical signs and symptoms	<ul style="list-style-type: none"> <li>Elevation</li> <li>Manual massage</li> <li>DEC</li> </ul>	Not required
Lymphoedema II <sup>1-4</sup>	Medical practitioner	Clinical signs and symptoms	<ul style="list-style-type: none"> <li>Limb hygiene, elevation, management of ADL</li> <li>Physiotherapy</li> </ul>	Not required
Lymphoedema III <sup>1-4</sup>	Medical practitioner	Clinical signs and symptoms	<ul style="list-style-type: none"> <li>Limb hygiene, elevation, management of ADL</li> <li>Physiotherapy</li> <li>Interferential therapy</li> </ul>	Not required
Lymphoedema IV <sup>1-4,12</sup>	Medical practitioner	Clinical signs and symptoms	<ul style="list-style-type: none"> <li>Topical application of antibiotics</li> <li>Interferential therapy</li> <li>Limb hygiene, elevation, management of ADL</li> <li>Physiotherapy</li> <li>Lymphonodal shunt</li> </ul>	Required when lymphonodal shunt is performed
Hydrocele (filaricele) <15 cm <sup>1-4</sup>	Trained surgeon	Clinical signs and symptoms	Surgery	Required
Hydrocele (filaricele) ≥15 cm <sup>1-4</sup>	Trained surgeon	Clinical signs and symptoms	Surgery	Required
Chyluria <sup>1-4</sup>	Medical practitioner	Urine test	Antibiotics to prevent secondary infection	Not required

ADL: adenolymphangitis; DEC: diethylcarbamazine; TPE: tropical pulmonary eosinophilia

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