



WEEKLY EPIDEMIOLOGICAL REPORT

A publication of the Epidemiology Unit
Ministry of Health

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Leishmaniasis

Background

Outbreaks of Leishmaniasis have been reported in Sri Lanka, mainly from Matara, Anuradhapura and Hambanthota districts. Most of these were cutaneous leishmaniasis, but the first case of visceral Leishmaniasis was reported from Anuradhapura in 2006.

Leishmaniasis comprises a complex of vector-borne diseases, caused by more than 20 species of the protozoan genus *Leishmania* and ranging from localized skin ulcers to lethal systemic disease. The most common syndrome is localized cutaneous leishmaniasis. Other forms of leishmaniasis are mucocutaneous leishmaniasis, disseminated cutaneous leishmaniasis, leishmaniasis recidivans and visceral leishmaniasis. Visceral leishmaniasis is a systemic disease, typical features of which include fever, wasting, splenomegaly, hepatomegaly and bone marrow suppression. Full blown clinical visceral leishmaniasis (sometimes called kala-azar) is lethal in nearly all untreated cases.

The Disease

Dozens of different sand fly vectors, adapted to different ecological settings where leishmaniasis occurs, are known to transmit some form of leishmaniasis. Leishmaniasis, especially the visceral form, can also be transmitted by blood transfusion or sharing of contaminated needles. Congenital transmission has been reported, but appears to be rare.

In sylvatic cycles, animal reservoir hosts can maintain transmission indefinitely without human disease. Sporadic or epidemic leishmaniasis occurs when humans enter the sylvatic habitat for economic or military purposes or when human habitation encroaches on the sylvatic setting. In domestic cycles, humans or dogs form the predominant or sole infection reservoir. In some areas, leishmaniasis transmission is zoonotic (dog-sand fly - human), while in other areas, it may be anthroponotic (human -sand fly -human).

The most common syndrome is localized cutaneous leishmaniasis, most frequently caused by *L. major* and *L. tropica* in the Old World (Europe, Asia and Africa) and *L. braziliensis*, *L. mexicana* and related species in the New World (Americas). The incubation period ranges from a few weeks to several years. The lesion may start as a nodule but eventually ulcerates in most cases. Growth is usually fairly gradual but continues over a period of months. In the absence of secondary bacterial infection, lesions are usually non-painful.

Local lymphadenopathy can occur, especially with species of the *L. braziliensis* complex. Spontaneous healing is common, but requires months to years. The proportion that heals without treatment and length of time required for healing vary by species.

Mucocutaneous leishmaniasis usually occurs months or years after healing of primary cutaneous leishmaniasis, most commonly due to parasites of the *L. braziliensis* complex and can cause destruction of the nasal septum, palate and other mucosal structures leading to devastating facial mutilation and rarely death from airway involvement. Factors associated with higher risk of mucocutaneous leishmaniasis include malnutrition and delay in treatment of the antecedent localized cutaneous lesion. Other complicated forms include disseminated cutaneous leishmaniasis (diffuse nodular non-ulcerating disease) and leishmaniasis recidivans (localized slowly progressive non-healing or relapsing lesions). These forms of the disease are rare, difficult to treat and can be severe.

Visceral leishmaniasis is usually caused by *L. donovani* and *L. infantum*. The time from an infective sand fly bite to the onset of visceral leishmaniasis is typically 2 to 6 months but can range from 2 weeks to more than 2 years. The onset is usually insidious with worsening symptoms over a period of weeks to months. The typical patient presents with fever lasting at least 2 weeks, malaise, weight loss and may complain of abdominal fullness related to organomegaly. Common clinical features include fever, wasting, splenomegaly, hepatomegaly, hypergammaglobulinemia and pancytopenia. Hepatomegaly is usually less prominent than splenomegaly. The course is complicated by immunosuppression and secondary bacterial infections, hemorrhage, anemia and when kala-azar occurs during pregnancy, foetal wastage or congenital leishmaniasis. Kala-azar is lethal in nearly all untreated cases. Even in treated patients, case-fatality rates are often 10% or higher. Jaundice, wasting, severe anemia and HIV co-infection are associated with increased risk of mortality. Post-kala-azar dermal leishmaniasis (PKDL) is a chronic rash seen in apparently cured kala-azar patients, presenting with erythematous or hypopigmented macules, sometimes progressing to plaques or nodules. Sometimes it resolves without treatment in most mild cases, while the condition is said to require treatment in some areas.

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Diagnosis

The diagnosis of cutaneous leishmaniasis relies on the demonstration of *Leishmania* in tissue biopsy, scraping or impression preparations by microscopy and/or culture in a specialized medium. Species identification is recommended because management may vary depending on the infecting species. Recently, assays based on the use of polymerase chain reaction (PCR), including multiplex assays that can distinguish among several species simultaneously, have become more widely available.

For visceral leishmaniasis, definitive diagnosis requires the demonstration of the parasite by smear or culture in tissue, usually bone marrow or spleen and thus entails an invasive procedure. Splenic aspirate has the highest sensitivity of available tissue sampling techniques, but carries a risk of serious hemorrhage. Bone marrow aspirates are safer, but have substantially lower sensitivity. Parasites can be detected in tissue samples by light microscopy of stained slides, culture in a specialized medium or by specific PCR assays. Serological tests (for example, immunofluorescent antibody tests or enzyme linked immunosorbent assays) can be used to demonstrate anti-leishmania antibodies. These assays have high sensitivity for visceral leishmaniasis in patients without HIV infection, but may show positive results due to subclinical infection or cross-reactions and are therefore less specific than tissue sampling. Sensitivity of serology was low among patients with HIV-visceral leishmaniasis co-infection. Therefore, parasitological diagnosis is advisable.

Treatment

Cutaneous leishmaniasis

Localized cutaneous leishmaniasis is not a life-threatening condition. Treatment decisions should take into account the degree of morbidity balanced against the potential side effects of therapy options. The choice of treatment is dependent on *Leishmania* species (especially for New World cutaneous leishmaniasis), number, size and location of lesions and the availability of specific treatment modalities.

Cutaneous leishmaniasis ulcers from several *Leishmania* species may heal without treatment, although healing usually takes months and will leave a scar. For one or a few small lesions not on the face or over a joint, careful follow-up without drug treatment may be appropriate. The exceptions are ulcers due to *L. braziliensis* and *L. panamensis*, which tend not to heal spontaneously and are associated with a small risk (<5%) of subsequent mucocutaneous leishmaniasis.

- The pentavalent antimonial drugs, sodium stibogluconate and meglumine antimoniate remain the most widely used anti-leishmanial agents, but are increasingly being replaced by safer drugs.
- Oral antifungal drugs (fluconazole, ketoconazole, itraconazole) have been used to treat cutaneous leishmaniasis with variable results depending on the *Leishmania* species and geographic location.
- Liposomal amphotericin B appears to have efficacy against several cutaneous leishmanial species, but data are limited and the optimal dose regimen has not been established.
- Other treatment modalities that have shown some efficacy for cutaneous leishmaniasis due to some *Leishmania* species include topical paromomycin ointment, oral miltefosine, thermotherapy and intralesional pentavalent antimonial drugs.

Visceral leishmaniasis (Kala-azar)

Patients with VL should be evaluated for HIV co-infection; if found, HIV should be treated aggressively. In the absence of treatment, the case-fatality rate of visceral leishmaniasis is >90%. Mortality is often due to hemorrhagic or infectious complications. Supportive therapy to address nutritional status, concomitant anemia, hemorrhagic complications and secondary infections is therefore essential to optimize treatment outcomes and maximize survival.

- Liposomal amphotericin B is the drug with the highest therapeutic efficacy and the most favourable safety profile
- The pentavalent antimonial drugs, sodium stibogluconate and meglumine antimoniate, remain the most widely used antileishmanial drugs.
- New drugs such as parenteral paromomycin and miltefosine are not readily available.

Prevention & Control

Prevention and control of leishmaniasis is based on two major modalities: to decrease human exposure to sand fly bites and interventions to decrease the infection reservoir of *Leishmania* species. However, the strategy for a given location must be tailored to the local epidemiology and ecology. Specifically, the best control modalities depend on the ecology of transmission (sylvatic or domestic), local sand fly behaviour (sylvatic versus peridomestic; resting in houses versus resting in outdoor niches); and the local reservoir hosts (wild animal, domestic animal or human).

Decreasing the Reservoir of Infection

For anthroponotic leishmaniasis, rapid diagnosis and effective treatment of patients decreases the infection reservoir. Theoretically, canine reservoir reduction should decrease transmission of zoonotic VL, but programmes that are focused on the culling of infected dogs have not proved highly effective, possibly due to inadequate sensitivity of diagnostic testing to detect infected dogs and time delays between diagnosis and culling. For sylvatic foci of cutaneous leishmaniasis, eliminating rodent reservoir hosts in a buffer zone around human dwellings may decrease transmission.

Vector Control

Indoor residual insecticide spraying (e.g. Pyrethroid insecticides) may be an effective intervention for sand fly vector species that rest inside human dwellings. Spraying usually needs to be repeated every 6 months. Use of insecticide-treated nets and other materials can be highly effective when the peak period of vector activity is late evening. Long-lasting insecticide-treated nets avoid the need for re-treatment, but the duration of their effect against sand flies under field conditions in endemic areas has not been evaluated. In areas of zoonotic leishmaniasis with a canine reservoir (e.g. *L. infantum*), treated dog collars and other modalities to decrease sand fly biting show promise as interventions to decrease infection exposure for humans.

The use of insect repellent and insecticide treated clothing is advisable for travellers, but is usually not feasible for the populations at highest risk.

Source

Leishmaniasis, available from

http://www.cdc.gov/parasites/leishmaniasis/health_professionals/index.html

Compiled by Dr. Madhava Gunasekera of the Epidemiology Unit

Table 1: Vaccine-preventable Diseases & AFP

31st December - 06th January 2012 (01st Week)

Disease	No. of Cases by Province									Number of cases during current week in 2012	Number of cases during same week in 2011	Total number of cases to date in 2012	Total number of cases to date in 2011	Difference between the number of cases to date in 2012 & 2011
	W	C	S	N	E	NW	NC	U	Sab					
Acute Flaccid Paralysis	00	00	00	00	00	00	00	00	00	00	01	00	01	- 100.0 %
Diphtheria	00	00	00	00	00	00	00	00	00	-	-	-	-	-
Measles	00	00	00	00	00	00	00	00	00	00	01	00	01	-100.0 %
Tetanus	00	00	00	00	00	00	00	00	00	00	01	00	01	-100.0 %
Whooping Cough	00	00	00	00	00	00	00	00	00	00	00	00	00	00 %
Tuberculosis	68	41	90	24	58	13	24	07	28	353	140	353	140	152.1 %

Table 2: Newly Introduced Notifiable Disease

31st December - 06th January 2012 (01st Week)

Disease	No. of Cases by Province									Number of cases during current week in 2012	Number of cases during same week in 2011	Total number of cases to date in 2012	Total number of cases to date in 2011	Difference between the number of cases to date in 2012 & 2011
	W	C	S	N	E	NW	NC	U	Sab					
Chickenpox	11	02	07	03	13	05	02	06	06	55	76	55	76	- 37.5 %
Meningitis	02 KL=2	00	00	00	00	08 KR=7 PU=1	01 AP=1	01 MO=1	01 RP=1	13	12	13	12	+ 8.3 %
Mumps	14	05	06	04	17	04	15	04	11	80	42	80	42	+ 90.5 %
Leishmaniasis	00	00	02 HB=2	00	00	00	04 AP=3 Po=1	00	00	06	01	06	01	+ 500.0 %

Key to Table 1 & 2

Provinces: W: Western, C: Central, S: Southern, N: North, E: East, NC: North Central, NW: North Western, U: Uva, Sab: Sabaragamuwa.
DPDHS Divisions: CB: Colombo, GM: Gampaha, KL: Kalutara, KD: Kandy, ML: Matale, NE: Nuwara Eliya, GL: Galle, HB: Hambantota, MT: Matara, JF: Jaffna, KN: Killinochchi, MN: Mannar, VA: Vavuniya, MU: Mullaitivu, BT: Batticaloa, AM: Ampara, TR: Trincomalee, KM: Kalmunai, KR: Kurunegala, PU: Puttalam, AP: Anuradhapura, PO: Polonnaruwa, BD: Badulla, MO: Moneragala, RP: Ratnapura, KG: Kegalle.

Data Sources:

Weekly Return of Communicable Diseases: Diphtheria, Measles, Tetanus, Whooping Cough, Chickenpox, Meningitis, Mumps.

Special Surveillance: Acute Flaccid Paralysis.

Leishmaniasis is notifiable only after the General Circular No: 02/102/2008 issued on 23 September 2008. .

Dengue Prevention and Control Health Messages

You have a duty and a responsibility in preventing dengue fever. Make sure that your environment is free from water collections where the dengue mosquito could breed.

Table 4: Selected notifiable diseases reported by Medical Officers of Health
31st December - 06th January 2012 (01st Week)

DPDHS Division	Dengue Fever / DHF*		Dysentery		Encephalitis		Enteric Fever		Food Poisoning		Leptospirosis		Typhus Fever		Viral Hepatitis		Human Rabies		Returns Received
	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	%
Colombo	219	219	3	3	0	0	9	9	0	0	2	2	0	0	0	0	0	0	92
Gampaha	117	117	3	3	0	0	1	1	0	0	3	3	0	0	6	6	0	0	73
Kalutara	36	36	6	6	0	0	3	3	0	0	5	5	0	0	1	1	0	0	85
Kandy	60	60	4	4	0	0	0	0	0	0	4	4	4	4	0	0	0	0	87
Matale	9	9	3	3	1	1	0	0	0	0	2	2	0	0	0	0	0	0	92
Nuwara	5	5	1	1	0	0	0	0	0	0	2	2	2	2	1	1	0	0	77
Galle	26	26	3	3	0	0	1	1	1	1	1	1	0	0	0	0	0	0	79
Hambantota	8	8	2	2	0	0	0	0	0	0	2	2	1	1	0	0	0	0	75
Matara	30	30	2	2	0	0	3	3	0	0	3	3	3	3	4	4	0	0	100
Jaffna	14	14	7	7	1	1	14	14	2	2	2	2	34	34	1	1	0	0	92
Kilinochchi	6	6	1	1	0	0	1	1	0	0	0	0	4	4	0	0	1	1	75
Mannar	15	15	1	1	0	0	2	2	0	0	1	1	0	0	0	0	0	0	100
Vavuniya	4	4	0	0	2	2	0	0	2	2	2	2	0	0	0	0	0	0	100
Mullaitivu	2	2	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	100
Batticaloa	40	40	4	4	0	0	1	1	0	0	0	0	0	0	0	0	0	0	86
Ampara	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	71
Trincomalee	11	11	4	4	0	0	2	2	0	0	0	0	0	0	0	0	0	0	83
Kurunegala	42	42	3	3	1	1	2	2	0	0	3	3	1	1	3	3	0	0	87
Puttalam	19	19	2	2	0	0	1	1	0	0	2	2	1	1	0	0	00	00	83
Anuradhapu	7	7	2	2	0	0	0	0	1	1	4	4	1	1	1	1	0	0	68
Polonnaruw	3	3	0	0	0	0	0	0	0	0	1	1	0	0	1	1	0	0	86
Badulla	5	5	2	2	0	0	1	1	0	0	0	0	0	0	1	1	0	0	76
Monaragala	5	5	3	3	1	1	2	2	0	0	7	7	0	0	1	1	1	1	82
Ratnapura	24	24	5	5	2	2	0	0	2	2	6	6	0	0	0	0	0	0	67
Kegalle	22	22	0	0	0	0	2	2	4	4	1	1	0	0	12	12	0	0	91
Kalmune	4	4	8	8	0	0	1	1	0	0	0	0	0	0	0	0	0	0	92
SRI LANKA	734	734	69	69	08	08	46	46	12	12	54	54	51	51	32	32	02	02	83

Source: Weekly Returns of Communicable Diseases WRCD).

*Dengue Fever / DHF refers to Dengue Fever / Dengue Haemorrhagic Fever.

**Timely refers to returns received on or before 06th January, 2012 Total number of reporting units 329. Number of reporting units data provided for the current week: 275

A = Cases reported during the current week. B = Cumulative cases for the year.

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Comments and contributions for publication in the WER Sri Lanka are welcome. However, the editor reserves the right to accept or reject items for publication. All correspondence should be mailed to The Editor, WER Sri Lanka, Epidemiological Unit, P.O. Box 1567, Colombo or sent by E-mail to chepid@sltnet.lk.

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