Leishmaniasis

By Jade Hecht

Transmission and Reservoir

The parasitic disease, leishmaniasis, is caused by protozoa of the *Leishmania* genus. Female phlebotomine sandflies carrying one of the twenty one species that infects humans can transmit this disease through their proboscis during a meal. When sandflies bite a human to eat, they inject the infectious stage of the protozoa, which is known as a promastigote. These promastigotes are then phagocytized by macrophages and other mononuclear phagocytic cells. Inside these cells, the promastigote transforms into the tissue stage of the parasite, known as an amastigote. The amastigotes go on to multiply and spread to infect other cells. When a sandfly bites an infected human, it ingests the macrophages that are infected with the amastigotes. Inside the fly’s gut, amastigotes transform into the promastigote stage. The parasite divides and migrates from the gut to the proboscis, and the cycle starts over.

Characteristics of *Leishmania*

The life cycle of *Leishmania* includes two hosts, a vertebrate and an invertebrate (i.e., the sandfly), making it a heteroxenous parasite. It is also digenetic, existing in two forms: the amastigote and the promastigote. As an amastigote, also called the Leishman-Donovan body, it infects the vertebrate intracellularly. In this form, the parasite is nonmotile and divides by longitudinal binary fission at 37°C. Although the word “amastigote” literally means “without flagellum,” the amastigote actually does have flagellum, but it is just not visible by light microscopy. As a promastigote, the flagellum extends from its body surface, making the flagellum easily visible under light microscopy. Ranging 15 to 30 µm in length, a promastigote is roughly five times longer than an amastigote. In the promastigote stage, *Leishmania* is motile and extracellular in the invertebrate. It also divides by longitudinal binary fission but at 27°C rather.

Tests and Identification

Testing for leishmaniasis can be done in a few ways in a laboratory. The first is directly examining a tissue sample for *Leishmania* under a light microscope. Another method is by the detection of the parasitic DNA by PCR and PCR ELISA. The final method is by the identification of the *Leishmania* antigen in tissue or fluid samples or the identification of “nonspecific or specific antileishmanial antibodies.”

Signs and Symptoms

There are many types of leishmaniasis, but the two most common are cutaneous leishmaniasis and visceral leishmaniasis. Generally, cutaneous leishmaniasis affects the skin and visceral leishmaniasis affects the internal organs. In cutaneous leishmaniasis, skin sores develop within a few weeks after being bitten from a sandfly. Skin sores can be either painful or painless, and they can start out as papules and nodules before progressing to ulcers. Visceral leishmaniasis usually affects the liver, spleen, and bone marrow, and it can be lethal. Visceral leishmaniasis takes longer to develop than cutaneous leishmaniasis, sometimes appearing after years. Signs and symptoms usually include fever, enlargement of the liver and spleen, weight loss, and low blood counts.
History

The history of leishmaniasis can be dated to the first century A.D. During this period, depictions of skin sores and deformities characteristic of cutaneous leishmaniasis and mucocutaneous leishmaniasis were found on pottery from Ecuador and Peru. Written observations by Incans and Spanish conquistadors from the 15th and 16th centuries recorded skin lesions on workers returning from the Andes. Because of these lesions’ resemblance to leprosy, leishmaniasis was called “white leprosy.” In the 18th century, visceral leishmaniasis was called “black fever” in Africa and India. The disease was eventually named its current name after William Leishman, a Scottish doctor, who made one of the earliest stains of Leishmania in 1901.

Virulence Factors

A factor in the virulence of Leishmania is the enzyme proteinase. This enzyme plays a large role in a protozoa’s migration through tissues, evasion of the immune system, degradation of hemoglobin, and activation of inflammation in mammalian hosts. The major surface protein, MSP or gp63, is a metalloproteinase and is commonly found on the surface of Leishmania and related protozoans. This protein aids in the protection of Leishmania from host enzymes and phagolysosomes of macrophages. Gp63 also interferes with “signaling cascades” and components of transcription, which can prevent the host cell from responding sufficiently to the parasite.

Control, Treatment, Prevention, and Vaccines

Fortunately, skin sores from cutaneous leishmaniasis usually go away on their own. Unfortunately, this can take months and sometimes even years, and it usually leaves behind scars. Visceral leishmaniasis, on the other hand, can be fatal if left untreated.

Drugs that treat visceral leishmaniasis include liposomal amphotericin B, which is administered intravenously, and miltefosine, which is administered orally. Miltefosine can also be used to treat cutaneous and mucosal leishmaniasis. Although they have been used to treat leishmaniasis since the 1940s, pentavalent antimonial compounds (Sb⁵⁺) are not available for commercial use in the U.S. Nevertheless, the Sb⁵⁺ compound sodium stibogluconate, Pentostam, is available to physicians through the CDC Drug Service.

The best thing is to prevent this disease altogether is by protecting oneself from the vector that transmits leishmaniasis, the sandfly. Wearing insect repellent, protective clothing, and limiting nighttime outdoor activity are all ways to prevent bites from sandflies. In endemic regions, spraying insecticides in homes and using mosquito nets around beds can also prevent the spread of leishmaniasis. There are currently no vaccines or drugs to prevent leishmaniasis.

Cases and Outbreaks

Leishmaniasis is found in almost 88 countries, mostly in warm tropical or desert regions. Some cases have emerged in Mexico and even Texas.

Globally, visceral leishmaniasis largely affects Brazil, India, Ethiopia, Somalia, Sudan, and South Sudan. 90% of all new cases that were reported to the World Health Organization in 2014 were in these six countries. An estimated 200,000 to 400,000 people are infected with visceral leishmaniasis each year. Most cases of cutaneous leishmaniasis occur in Afghanistan, Saudi Arabia, Iran, Pakistan, Peru,
Brazil, Colombia, and Algeria. Estimates of cutaneous leishmaniasis cases range from 700,000 to 1.2 million per year.

A few incidences of cutaneous leishmaniasis have been reported in Texas and Oklahoma. Usually these cases are the result of travel, especially popular tourist destinations like Costa Rica.

References