American Visceral Leishmaniasis in Colombia

The first case of American visceral leishmaniasis (AVL) known in Colombia was described by Gast Galvis to the National Academy of Medicine in 1944, and consisted of a finding in viscerotomy material from the Department of Santander. Later (1968) three cases of the disease were reported in the same Department (Gómez Vargas), and three more in the Departments of Santander and Cundinamarca (Castillo *et al.).*

Epidemiological studies in 1971 at Melgar and Coyaima, Department of Tolima (Corredor, Osorno, and Parra) revealed the following:

- The phlebotomines were represented chiefly by *Lutzomyia longipalpis* (mosquito) as in the foci studied by other workers in the Departments of Cundinamarca, Huila, and Tolima; and secondarily by *L. trinidadensis, L. rangeliana, L. gomezi,* and *L. cayennensis.*
- *L. longipalpis* was always found in the hollows of rocks, in shelters adjacent to dwellings, and was not seen at altitudes above 900 m.
- In El Aguila (Melgar), 15% of the dogs were infected, and in that of Santa Marta (Coyaima), 3.8%.
- In Santa Marta 10.6% of the human population showed titers above 1:20 by the complement-fixation test using BCG antigen. Reactions to the complement-fixation test for Chagas' disease were obtained for 25% of the population.

The antigen used in the complement-fixation test was the ketone extract of the BCG tubercle bacillus or of *Mycobacterium butyricum.* The test is highly sensitive, yielding results in cases that have been progressing for less than three months. It also gives cross reaction with leprosy, tuberculosis, Chagas' disease, and tegumentary leishmaniasis.

In Colombia AVL foci are located in areas in the consolidation phase of the malaria eradication campaign (Figure 1). This bears out the assumption that the disease has existed for years in that country, and its diagnosis must have long been masked by the visceral forms of malaria. It has become possible to diagnose cases of AVL more frequently in malarious areas in which the frequency of malaria has been reduced.

During the four years (1958-1962) of the attack phase of the antimalaria campaign using DDT spraying, the peridomestic transmission of leishmaniasis ceased, but a cycle of transmission continued among foxes and other animals in the wild. Within an indeterminate period following the suspension of spraying in October 1962, peridomestic transmission resumed, and canine and human cases reappeared.

The masking of this entity by malaria and the fact that it is not regarded as a diagnostic option suggest that many patients die of this pathology as the result of erroneous diagnoses.

A compilation of the cases on record from 1944 to 1980 by Corredor, Ronderos, and Rey contains 107 cases verified by clinical examination and by smear or culture in the laboratory (Table 1). The rural distribution of the disease covers the Upper Magdalena River Valley, the principal focus spanning the border areas between the Departments of Cundinamarca and Tolima and between Tolima and Huila, the central part of the Department of Santander, and Ovejas Municipality in the northwestern Department of Sucre.

What follows refers only to the five-year period 1976-1980, for which the available information is more complete. There are no exact data on the population exposed to AVL, since the foci discovered are in rural populations with no census. Moreover, other foci are believed to exist, but have not been identified owing to the limitations stated above.

Figure 1. Visceral leishmaniasis foci in Colombia, 1944-1980.

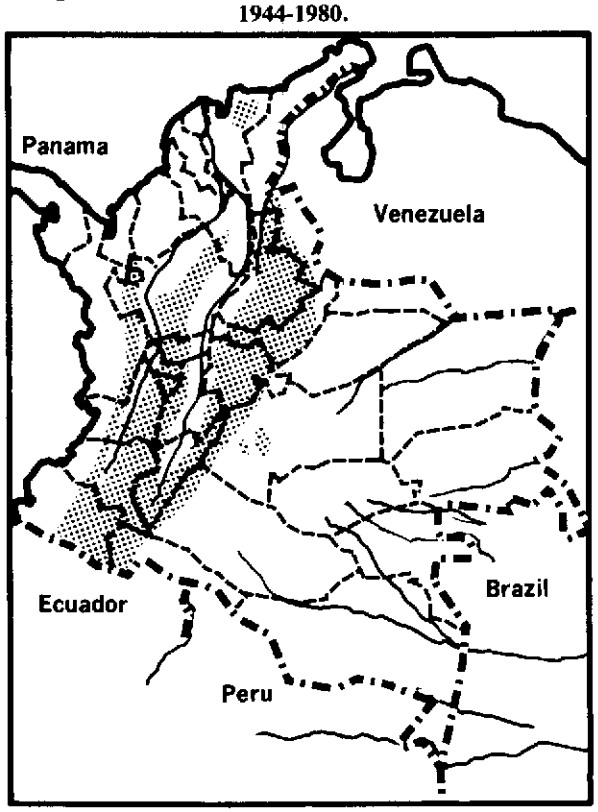

Table 1. Distribution of American visceral leishmaniasis by age groups and place of origin, Colombia, 1944-1980.

Place of origin	Age groups				%	Total
	-1	1-4	5-14	15+		
Tolima	10	32	6	1	45.8	49
Cundinamarca	7	15	3	3	26.2	28
Huila	2	15	3	1	19.6	21
Santander	-	2	2	2	5.6	6
Sucre	-	2	1	-	2.8	3
Total	19	66	15	7	100.0	107

In recent years an increase in AVL reporting has been observed in Colombia. This is probably because of the launching of the specific case-finding program which has increased the availability of therapeutic care at the regional level, medical and paramedical education on the salient characteristics of the disease, and the dissemination of educational information in the communities, resulting in increased numbers of early diagnoses.

The hardest hit group during the five-year period (1976-1980) was that of children under five, who accounted for 86.5% of all cases. In this group, the incidence among infants under one year (1.75/100,000 inhabitants) exceeds the incidence among children 1-4 years (1.67/100,000 inhabitants) even though in terms of absolute numbers the reverse may appear to be the case. Since in these rates the denominator is the population at large and not the population at risk (that living in rural areas at an altitude below 900 m, where the vector is present), the specific rates are presumably much higher and point to a public health problem in the endemic areas.

The fact that children under five years of age are the most severely affected group is in line with the pattern in Brazil, Venezuela, and the subtropical zone. Several explanations for this have been outlined. One is that immunity to AVL correlates directly with the time of exposure, which would imply a high probability of immunity in adults and greater susceptibility to the disease in children.

It could also be that *Leishmania infantum* of the Mediterranean region and *L. chagasi* of the Americas strike children under five because of some characteristic in children that makes them susceptible to the infection, unlike *L. donovani*, which attacks individuals of all ages. Moreover, the infectivity of *L. chagasi* depends on the parasite strain, the susceptibility of the host, and other vector-related mechanisms.

It would seem that under natural conditions not all infections progress toward a clinical picture of visceral leishmanisis. Some cases are benign, asymptomatic, and result in spontaneous recovery yet acquire immunity.

In leishmaniasis, sterilizing immunity is imparted by cell-mediated reactions. From the appearance of the very first symptoms of the infection, leishmania antibodies can be detected. As the disease progresses, unspecific antibodies increase greatly along with specific ones, and are accompanied by a rise in gamma globulins and a reduction of albumins. Humoral antibodies do not sterilize, and the cure is concurrent with the appearance of cell immunity, delayed hypersensitivity, and resistance to reinfection.

(Source: Parasitology Section, National Institute of Health, Ministry of Health, Colombia.)

Editorial Comment

American visceral leishmaniasis is a generalized chronic infectious disease characterized by fever, hepatosplenomegaly, lymphadenopathy, anemia with leukopenia and progressive weakness, which produces high mortality among children. It is a zoonosis of canines, both wild (foxes) and domesticated (dogs), in which the infection is transmitted from animal to animal by *Lutzomyia longipalpis*, which bites both foxes, and dogs and man.

Man is an occasional host when he enters the vector's food chain. The leishmanias that cause visceral leishmaniasis are currently classed in three well-defined groups: *Leishmania donovani* (Laveran and Mesnil, 1903), *L. infantum* (Nicolle, 1909), and *L. chagasi* (Marqués de Cunha and Chagas, 1937).

This protozoosis was first diagnosed in 1913 by Migone in Paraguay in a patient from the State of Mato Grosso, Brazil. Since then it has been recorded in equatorial, tropical, and subtropical climates of the Americas, specifically in Argentina, Bolivia, Brazil, Colombia, Ecuador, El Salvador, French Guiana, Guatemala, Honduras, Mexico, Suriname, Paraguay, and Venezuela. Incidence is greatest in rural areas and in the peripheral sections of cities that retain certain rural features.

Identified AVL foci are located in mountainous regions at altitudes not usually above 800 m. The topography in these areas is generally rugged—valleys alternating with mountain spurs—and the climate is dry or moderately humid. These topographical features char-

acterize foci described in Brazil, Venezuela, Central America, and Colombia.

For proper control of the disease, countries with areas in which AVL is endemic should encourage the following activities:

Diagnosis. To the extent possible, the use of the fluorescent antibody technique (FAT) should be adopted as a standard procedure. Other serologic techniques, such as the enzyme-linked immunosorbent assay (ELISA), can be introduced.

Information Analysis. There must be increased epidemiological analysis and interpretation of collected data, with every effort made to avoid duplicate records. Analysis must emphasize the trend of the disease over time for places of incidence, most severely affected age groups and sex, social factors involved, etc. Maps should also be prepared showing the geographic distribution of the endemic foci.

Antibody Levels. Some positive cases can be monitored clinically and serologically to determine the levels and duration of specific post-treatment antibodies in the serum.

Special Epidemiological Studies. Serologic studies using the FAT or ELISA should be done to evaluate the prevalence and distribution of infection. These data can be entered on maps of irrigation zones, land use, and phytogeography.

Search for Reservoirs: It is recommended that the following studies be attempted:

- a study of the flora and fauna in the endemic area,
- special serologic and parasitologic studies in stray dogs to find signs of infection,
- examination of foxes and rodents in endemic and adjacent areas, and
- isolation and characterization of the leishmania in these animals.

Entomologic Studies. A well-conducted entomologic study should be done in an attempt to distinguish the vectoral densities of the different species of phlebotomi, their geographic and seasonal distributions, the preferred host of each species, and the vectoral capacity.

Vaccines: The Way Ahead[1]

Communicable diseases caused by parasites, bacteria, and viruses continue in the 1980s to impose a major burden of morbidity, mortality, and disability on the world's populations. The greatest hope of reducing this toll lies in immunization.

Acute diarrheal diseases are widespread and are the cause of more than 4.5 million deaths a year in children less than six years old; acute respiratory infections cause over 2 million deaths a year; while malaria, schistosomiasis, and other tropical diseases in warmer climates and tuberculosis—mainly in poorer areas—are major scourges.

Future improvements in environmental and nutritional standards may reduce the incidence and severity of some of these diseases. New drugs and antibiotics for prophylaxis and therapy will, of course, continue to be developed, but many will lose their efficacy as the infecting organisms become resistant.

Passive immunity, conferred by the injection of preformed specific antibody, can last only while the antibody remains in the recipient. Such therapy or prophylaxis is expensive and brings its own range of undesirable side effects and is therefore not envisaged for general use.

In contrast, vaccination against viral and bacterial agents appears to be of wide applicability and is generally a safe procedure. Once economical methods are developed for their preparation, vaccines may be made in large quantities and usually cheaply. For example, trivalent live poliovaccine may cost a fraction of a dollar per dose. Immunity induced by one to three doses of vaccine is generally long lasting. Vaccination against parasitic diseases has still to be fully developed, but recent vaccines against malaria show considerable promise.

Mankind is now on the threshold of a new era in the technology of vaccine development and production, which stems from important advances in biotechnology, in particular recombinant DNA and cell fusion techniques. It offers hope of producing vaccines for many of the diseases that are yet uncontrolled and also of developing vaccines that are more effective, safer, and more cost-effective than those in current use.

[1]Reprinted from *World Health Forum* 4(4):408-413, 1983.