Emerging and Reemerging Infectious Diseases

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Objectives

By the end of this educational encounter the clinician will be able to:

1. Identify reasons that the clinician may encounter an emerging or reemerging infectious disease.
2. Identify diseases that the clinician is likely to encounter
3. Identify characteristics and treatment for diseases

The purpose of this educational activity is to acquaint the clinician with current emerging and reemerging infectious diseases that could feasibly be encountered in the clinical setting.

Introduction

Health experts are expressing concern that the United States may begin to see cases of new or reemerging diseases, and some of those diseases may seem unusual in the fact that they are thought to be tropical or exotic in nature. However, in today's very mobile world the health care community is no longer immune to disease because of distance. We are now living in a global environment in which we must begin to think of healthcare in terms of the world in general, not just the bounds of the continent upon which we live. With international air travel more common, it is possible for any disease or virus to be transported from its point of origin to the North American continent within a 24-hour period of time.

In addition to global travel there are many other reasons that are contributing to the spread of emerging and reemerging infectious diseases.

- Poverty is a major factor in the spread of disease. When a person lives in poverty, they are more likely to live in crowded, unsanitary conditions. Living without electricity, running water, and proper sewage disposal sets the stage for a hospitable breeding ground for many infections. Close physical contact

due to overcrowding leads to increased risk for skin and parasitic infestations such as fungal infections and lice.

- Persons living in poverty are likely to lack health insurance and not receive access to needed healthcare and preventative health programs.
- Social disruption leads to the spread of disease through the fleeing of persons due to war, political strife, or natural disasters.
- Personal health behaviors such as drug use, food handling and preparation procedures, risky sexual behavior, hand hygiene habits, or failure to follow preventative health procedures such as vaccination programs.
- Increased susceptibility due to immune system disturbances, medical treatments, and increased age or chronic disease.
- Breakdown of public health infrastructures
- Change and evolution in microbes
- Global commerce, including the trade and import/export of animals and foodstuffs
- Economic growth can changes in the use of land
- Climate and weather conditions, including global warming.
- Disruption in ecosystems due to farming, forest clearing, and reclamation
- Technological changes in industry, and healthcare
- Lack of political will [1]

Chagas Disease

Chagas disease, also known as American trypanosomiasis is a parasitic protozoan infection caused by infection with the Trypanosoma cruzi parasite. T. Cruzi is found mostly in blood sucking insects and small mammals. The main areas of disease are found in the southern and southwestern United States down to Argentina and Chile. Transmission occurs through contact of infected droppings with breaks in the skin or mucous membranes. Transmission can also be through congenital means or by blood transfusions or organ transplants. Transmission has also been documented by the consumption of contaminated foodstuffs, notably the acai fruit.

Infection with the T. cruzi is life-long. Inflammatory lesions that may appear at the site of entry are called chagomas. The adjacent lymph nodes may swell and enlarge. Acute cardiac involvement, myocarditis, may develop and consist of patchy areas of necrosis and infected cells.

Elevated lymphocyte and transaminase levels may be noted. Parasites may be found in the cerebrospinal fluid. The heart is the most frequently affected organ with ventricular enlargement being commonly seen. The right side is more often affected than the left. The walls of the ventricles are often thin, with mural clots and aneurysms present. Dysrhythmia may be seen due to conduction defects originating from fibrotic lesions in the bundle of His. Involvement of the gastrointestinal system is most

frequently manifested as dilation and hypertrophy of the colon or esophagus. Fibrosis is present and the number of neurons is frequently diminished.

There are 80,000-120,000 cases of Chagas disease in the United States. However most of these cases come from the fact that many immigrants with the disease have taken up residence in the United States. In the U.S., only one in 29,000 blood donors is infected with Chagas disease, however in Mexico only 13% of all blood donors are screened for the illness.

Acute Phase

The incubation period for acute chagas disease is 7-14 days but is not definite due to the fact that persons experiencing acute illness are most frequently still at risk for exposure to the carriers of the illness. Symptoms of acute Chagas disease may include malaise, anorexia, myalgia, and headache, but many recently infected persons are asymptomatic. Sporadic fevers also occur. Most persons who are acutely infected with the disease are not diagnosed due to the non-specific nature of the symptoms and the disease occurs most frequently in persons having limited access to medical care.

> ”Some patients have lesions at the portal of entry of the parasites. Romaña sign occurs when the parasites have contaminated the conjunctivae. Romaña sign is viewed as a classic sign of acute Chagas disease but develops in few newly infected persons. A chagoma, which is an indurated inflammatory skin lesion, may develop when parasites enter through a break in the skin. Romaña sign and chagomas may persist for several weeks. The lymph nodes that drain either of these lesions may be enlarged.” [2]

The mortality rate of the acute phase of the disease is estimated to be less than five percent, but this figure is not verifiable mainly because the disease is so seldom identified in the acute phase. When death does occur is most often likely due to cardiac complications.

In most persons with acute Chagas disease, the symptoms resolve within 3-8 weeks, which is then followed by the chronic phase or the latent phase (symptom free) of the illness.

Indeterminate Phase

According to true definition, the indeterminate phase of the illness has no symptoms. Most adults are unaware of their illness and are unable to report a history of any symptoms. Persons who are diagnosed at this stage of illness are identified through routine blood donor testing or routine employment serology testing.

Chronic Phase of the Illness

Between 10 to 30 percent of all persons that have chronic Chagas disease have clinical symptoms of the illness. The most serious of the manifestations are cardiac in nature and are caused by the presence of parasites within the heart, leading to inflammation of the heart muscle and cardiopathy. Peripheral edema, liver enlargement, ascites, and pulmonary edema are common in the latter stages of the illness.

The multidimensional cardiac symptoms manifested in chronic Chagas disease include:

- A wide array of atrial and ventricular rhythm malfunctions such as right bundle branch block, left anterior hemiblock, and third degree AV block. These disorders can lead to hypotension, palpitations, syncope, dizziness, and even sudden cardiac death.
- The cardiomyopathy and resultant heart failure can manifest symptoms such as shortness of breath, low exercise tolerance, peripheral edema, frequent urination at night, and extreme fatigue.
- Tromboembolism can cause stroke, as well as thrombi to the pulmonary and arterial circulation.

Gastrointestinal symptoms of Chagas disease most frequently occur as a result of denervation of the intestinal system and the resultant dysfunction.

- Megaesophagus is the most common gastrointestinal manifestation and effects are difficulty swallowing, substernal discomfort, and the sensation that foods and liquids are becoming stuck in the throat or do not go all the way down properly. Weight loss and a generalized failure to thrive may be present. The salivary glands are enlarged and pneumonitis is commonly seen due to the nighttime regurgitation and aspiration of stomach contents. The erosive esophagitis that accompanies the regurgitation puts the patient at increased risk for esophageal cancer.
- Megacolon causes constipation and pain, with affected patients sometimes going for long periods between bowel movements. In cases where the symptoms have remained for a protracted period, bowel obstruction requiring surgical intervention has occurred. Abdominal distention is often a feature and fecaloma can be present.
- Gastric dilation, in addition to megaureter, has been noted but these have been rare findings for this illness.

Treatment

The individual manifestations of the illness are treated conventionally, and parasites are eliminated, if appropriate, using benznidazole and nifurtimox. Surgical care is used when called for to treat cardiac, and gastrointestinal symptoms. Diet consists of foods appropriate in the treatment of congestive heart failure patients. Warm, pasty foods and small volumes of water will assist patients who have difficulty swallowing. Patients should allow plenty of time between eating and lying down to allow for digestion and prevent regurgitation. The diet should also include high fiber foods for those individuals that have intestinal symptoms. [2]

Malaria

Malaria is a mosquito-borne disease that causes over 2. 7 million deaths per year according to estimates by the World Health Organization. About 1,300 cases of malaria are diagnosed in the United States each year. The vast majority of cases in the United States are in travelers and immigrants returning from malaria-risk areas, many from sub-Saharan Africa and south Asia.

Malaria is a life-threatening disease caused by parasites that are transmitted to people through the bites of infected mosquitoes, the parasite then in turn infects red blood cells. A malaria infection starts about 9 to 14 days after being bitten. These parasites, because of their large numbers, can cause particular damage to the nervous system, liver, and kidneys. Malaria is characterized by cycles of chills fever, diarrhea, and sweating. Toxins released when the red cells burst are what causes the typical fever, chills and flu-like symptoms. Attacks can sometimes become severe, leading to serious anemia, convulsions, coma, and even death. Young children and pregnant women are particularly at risk of developing severe malaria. In cerebral malaria, the infected red cells obstruct the blood vessels in the brain. Other vital organs can also be damaged.

A traveler's best defense against this disease is to take antimalarial drugs and avoid being bitten by mosquitoes. They should remain in well-screened areas from dusk to dawn, sleep under mosquito netting, wear pants and long sleeved shirts, and apply mosquito repellent containing deet to their clothing and exposed skin. Reviews of travelers returning from endemic areas have reported that P falciparum infection typically develops within one month of exposure, thereby establishing the basis for continuing antimalarial prophylaxis for 4 weeks upon return from an endemic area.

Severe manifestations of malaria are:

- Cerebral malaria is almost always caused by infection with the P falciparum form of the disease. Coma may occur and can be distinguished from a postictal state

due to generalized seizure when the patient fails to regain consciousness within 30 minutes.

- Severe anemia is associated with malaria and may be multifactoral. P falciparum is the usual causative parasite. Anemia may be due to the loss of infected red blood cells. Bone marrow suppression may be present
- While a rare manifestation of malaria, renal failure does occur as a result of RBC's lodging to the minute blood vessels of the renal cortex. This causes renal failure and diminished urine output to the point that oliguria is present. With supportive dialysis, the renal failure may be reversible. In rare cases the renal failure is permanent.
- Patients with malaria may develop metabolic acidosis and respiratory distress. Pulmonary edema may be present. Patients may have nasal flaring, sternal retractions, abnormally deep breathing and accessory muscle use.

Treatment

When treating malaria it is very important to determine the species of the parasite involved. This may be done by blood smear. Infection with P falciparum may be more severe than infection with other Plasmodium species. This species may be resistant to chloroquine treatment. In the United States, patients with this type of infection are often admitted to the hospital to observe for complications directly related to the illness or its treatment.

Consultations

Patients with malaria should receive a consultation from an infectious disease specialist for assistance with diagnosing, determining the species of infection, treating the patient, and managing the disease. If no infectious disease specialist is available, the CDC is in excellent resource. The mall area hot line is available to speak with a phone call malaria specialist; the telephone number is 770-448-7788 or 770-488-7100.

Diet

Malaria patients can continue to eat and drink as tolerated.

Activity

There are no restrictions on the activity for malaria patients. They may continue with activity as tolerated.

"The following is a summary of general recommendations for the treatment of malaria:

- P falciparum malaria

    - Quinine-based therapy - Quinine (or quinidine) sulfate plus doxycycline or clindamycin or pyrimethamine-sulfadoxine
    - Alternative therapy - Atovaquone-proguanil or mefloquine
- P falciparum malaria with known chloroquine susceptibility (only a few areas in Central America and the Middle East) - Chloroquine
- P vivax, P ovale malaria - Chloroquine plus primaquine
- P malariae malaria - Chloroquine
- P knowlesi malaria – Recommendations same as those for P falciparum malaria" [3]

Dengue

Dengue is the most common arboviral illness that is transmitted the worldwide. There are four serotypes of the Dengue virus that are transmitted by mosquitoes which are found widely in the tropical and subtropical areas of the world. Dengue is classified as a major health threat by the World Health Organization.

Initially, dengue infection may be without symptoms, or may produce the classical symptoms of dengue fever. Dengue hemorrhagic fever results when a person previously infected with one type of the virus becomes infected with another type of the virus. Those persons develop bleeding and endothelial leak. Some patients with dengue hemorrhagic fever develop dengue shock syndrome, which can be fatal.

The virus transmission follows two general patterns -epidemic dengue and hyperendemic dengue. Epidemic dengue transmission occurs when dengue virus that comes introduced to a region in an isolated event that involves a single strain of the virus. If the number of hosts is sufficient over whelming transmission can occur with an infection rate of up to 50%.

Hyperendemic dengue transmission is distinguished a continuous circulation of multiple types of the virus in areas where large amounts of susceptible hosts are continually present. This is the usual pattern of global transmission. In these populations, antibodies increase with age making most adults immune. Hyperendemic transmission seems to be a major risk factor for dengue hemorrhagic fever. Travelers to hyperendemic areas run a higher risk of being infected vs. travelers to areas that experience only epidemic transmission.

Dengue fever is widespread across the globe; the only continents that do not have transmission of the virus include Europe and Antarctica. Dengue is found in many popular tourist countries including South America and the U.S. Virgin Islands. Two of the mosquitoes that are responsible for Dengue transmission are seasonally abundant in the United States including the states of Texas, Arizona, New Mexico, Louisiana, Mississippi, Alabama, Georgia, and to mid South Florida. There have

also been sporadic reports in portions of North Carolina, South Carolina, Tennessee, Arkansas, Maryland, and New Jersey. The range of one mosquito vector expands almost as far north as the Great Lakes.

Once bitten by an infected mosquito the incubation period for the dengue virus is 3 to 14 days. After incubation a 5 to 7 day acute febrile illness begins. The patient usually recovers within 7 to 10 days. When dengue hemorrhagic fever or dengue shock syndrome develops it usually begins around the third to seventh day of illness. Plasma leakage and bleeding are the major abnormalities characteristic of dengue hemorrhagic fever. Plasma leakage is caused by increased capillary permeability and may appear as hemoconcentration, pleural effusion, or ascites. Capillary fragility and thrombocytopenia cause bleeding in many forms including skin hemorrhages and profuse life-threatening GI bleeding.

Mortality/Morbidity

- Recovery from dengue infection is usually complete. Even patients who have had dengue hemorrhagic fever or dengue shock syndrome usually make a complete recovery.
- The fatality rate for dengue shock syndrome varies between 12 to 44%. The mortality rate for dengue fever is less than 1% .For every clinically apparent case of dengue fever 14 cases go unrecognized.
- Disease severity is affected by several factors patient's age, nutritional status, race, type of virus, and the available medical care.

History

Persons with symptomatic dengue fever may have fever as high as 105.8°Fahrenheit. Fever usually begins on the third day and lasts 5 to 7 days. Facial flushing, skin mottling, and chills are usually present prior to the start of the fever. Sometimes saddleback fever will be present in which the fever abates for a day, only to return.

Generalized headaches are often present with pain behind the eye orbits that is often described as severe. Nausea and vomiting may also exist. A maculopapular rash (present in approximately half of dengue patients) typically begins on day 3 with a 2-3 day duration and is manifested over the face, thorax, and flexor surfaces interspersed with rash free islands of skin.

The patient may complain of severe muscle and joint pain especially in the lower back, the extremities. Hemorrhage may be manifested as small amounts of bleeding from the nose or mouth to larger volumes of blood in the form of menstrual losses or gastrointestinal melena or hematemesis.

Patients who develop dengue hemorrhagic fever may initially be restless with a diminished mental status and complain of abdominal pain. Patients will have subnormal body temperatures and the platelet count will diminish. Sore throat, fatigue and cough are frequently reported.

Tachycardia is usually present during the febrile period, with the pulse becoming bradycardiac after the fever has passed. Conduction defects are not uncommon.

Generalized lymph node involvement frequently occurs and the liver may possibly be enlarges although this happens more frequently in dengue shock syndrome. Liver enzymes may be elevated.

Dengue shock syndrome is characterized by a rapid, but weak pulse, a pulse pressure of less than 20 mm Hg, hypotension, cold clammy skin, and altered mental status.

Treatment

There is no specific treatment for patients with Dengue virus; the care is usually focused at symptom management, fluids and volume expanders and monitoring for bleeding. Diet is as tolerated and bed rest is usually indicated in all cases [4].

Leishmaniasis

Leishmaniasis is a type of protozoal disease that can cause a spectrum of disease manifestations ranging from skin ulcerations to systemic manifestations. The parasites have been found throughout the world with exception of Australia, Antarctica, and Pacific Islands.

The parasites are transmitted by the bite of the female sand fly. In most instances, animal reservoirs must be present for the disease to be endemic. Humans are considered to be incidental hosts. Infections in wild animals are usually not pathogenic; however, dogs may be severely infected. In addition to dogs, other common hosts include sloths, anteaters, opossums, and rodents.

While the disease is not common in the United States, cases are increasing due to international travel, immigration, and the prevalence of United States Military personnel serving overseas. Epidemic currently exist in Afghanistan, Brazil, India, and Sudan. Since 2001 700 cases of leishmaniasis have been diagnosed in US Military personnel. Most of the cases in the United States have been reported in Texas and Southern Oklahoma.

Female sand flies can transmit the parasite 7-10 days after feeding on an infected host. After inoculation, parasites infect the reticuloendothelial system and live inside the cell

of macrophages. Parasites may incubate for weeks to months before presenting as skin lesions or as infection involving the liver, spleen, and bone marrow. In India, visceral leishmaniasis does not seem to have an animal reservoir and is thought to be transmitted via human-sand fly-human interaction.

The extent and manifestation of the disease depends on several factors, including the immune response of the host, the virulence of the infecting species, and the parasite burden. Infections may heal on their own or may progress to chronic disease, often resulting in death from secondary infection.

Mortality/Morbidity

Localized cutaneous leishmaniasis often resolves on its own within 3-6 months without any treatment; however some infections may persist for an indefinite period. Cases of diffuse cutaneous leishmaniasis, post-kala-azar dermal leishmaniasis, and leishmaniasis recidivans are usually chronic in nature and resistant to treatment, but are associated with low mortality rates.

Mucocutaneous leshmaniasis is chronic and can lead to death when the mucosa of the respiratory tract is invaded. The resulting dysphagia and respiratory compromise can cause pneumonia and malnutrition. The disease is usually progressive.

Visceral leishmaniasis is progressive and carries a mortality rate of 75-95% if the disease is untreated. With adequate treatment the death rate falls to around 5%. Death is usually from secondary infections and/or malnutrition.

Males tend to become infected more than females due to occupational and leisure exposure to the areas where the sandfly habitates.

History

Cutaneous leishmaniasis

Incubation occurs over weeks to months followed by the appearance of a reddened papule, which can evolve into a plaque or ulcer, which can spread over time.. Healing may occur spontaneously from 2-12 months and is followed by scarring and changes in pigmentation. Systemic symptoms are absent.

Lesions are usually found in exposed areas without pain or itching, however subsequent infection may complicate the wound. New World disease usually presents with a single nodule, and may progress to mucocutaneous leishmaniasis. While Old World disease is associated with multiple lesions. The border often has a raised erythematous rim known as the volcano sign, leaving a characteristic retracted hypopigmented scar.

Widespread cutaneous leishmaniasis develops in an immunocompromised host. Infection is characterized by a primary lesion, which spreads to involve multiple areas of the skin, which may form over the entire body, resembling lepromatous leprosy. There is no neurologic or systemic involvement. Infections are chronic, may be resistant to therapy and may recur despite treatment.

Leishmaniasis recidivans may occur years after healing of a localized skin lesion, commonly presenting on the face. New lesions form over the edge of the old scar and proceed inward. Infection may be from reactivation of dormant parasites or new infection from a different species. Infections tend to be resistant to treatment.

Post-kala-azar dermal leishmaniasis has predominantly been described in Africa and India. The Indian version occurs several years after recovery from visceral leishmaniasis and is characterized by multiple lesions. Over time, these can progress into large plaques and nodules that involve the face and trunk requiring intensive therapy. The African variant occurs shortly after treatment of visceral leishmaniasis and is characterized by multiple papular lesions of the face, buttocks, and extremities which spontaneously resolve over the course of several months.

Mucocutaneous leishmaniasis

Mucocutaneous disease is most commonly caused by New World species. A persistent skin lesion that eventually heals characterizes initial infection. Several years later, oral and respiratory involvement occurs, causing inflammation and damage to the nose, mouth, pharynx, and trachea. Patients may have rhinorrhea, epistaxis, and nasal congestion.

Examination reveals excessive tissue obstructing the nares, septal granulation, and perforation. Nose cartilage may be involved, giving rise to external changes known as parrot's beak or camel's nose. Progressive disease is difficult to treat and often recurs. The palate, uvula, lips, pharynx, and larynx may exhibit granulation, erosion, and ulceration with sparing of the bony structures. Hoarseness may be a sign of laryngeal involvement. With prolonged infection, death occurs from respiratory compromise and malnutrition.

Other physical signs include gingivitis, periodontitis, and localized lymphadenopathy. In time, disfiguring facial deformities may occur, requiring plastic surgery. Optical and genital mucosal involvement has been reported in severe cases.

Visceral leishmaniasis

Visceral disease, the most devastating and fatal form, is classically known as kala-azar or black fever. It occurs with both New and Old World species and results from systemic infection of the liver, spleen, and bone marrow. The syndrome is characterized by fever, weight loss, night sweats, diarrhea, enlargement of the liver and spleen, decreases in red and white blood cells, and increased immunoglobulins.

Physical examination reveals a patient who is thin and cachetic with abdominal distension and protuberance. The liver and spleen are usually soft and easily palpated, and the patient may experience intermittent abdominal distress. Epistaxis and petechiae from severe thrombocytopenia may occur. Lymphadenopathy and hair changes, such as alopecia and eyelash elongation, may be present. Characteristic patchy darkening of the face and trunk has been described. Complications of visceral leishmaniasis include amyloidosis, glomerulonephritis, and cirrhosis. If left untreated, death frequently occurs from immunosuppression and secondary infections.

Viscerotropic leishmaniasis

Viscerotropic disease has been described in patients returning from the Middle East. Symptoms emerge anywhere from 1 month to 2 years after exposure with malaise, fatigue, intermittent fever, cough, diarrhea, abdominal pain, and other gastrointestinal symptoms.

Viscerotropic leishmaniasis has a distinct clinical presentation and does not appear to progress to full visceral leishmaniasis.

Diagnosis and Treatment

Historically, the diagnosis of leishmaniasis has been confirmed by identifying the parasite in infected tissue. Significant advances in polymerase chain reaction (PCR) techniques have allowed for the highly sensitive and rapid diagnosis of specific Leishmania species. Although currently limited to military and reference laboratories, leishmania PCR diagnosis is becoming more widely available in developing-world laboratories and field sites.

Serological detection of antibodies to recombinant K39 antigen using a direct agglutination test, immunofluorescence assay, or enzyme-linked immunosorbent assay (ELISA) have been shown to be highly sensitive and specific in diagnosing visceral leishmaniasis.

Cutaneous and mucocutaneous forms generally display normal laboratory values.

For cutaneous leishmaniasis, a punch or wedge biopsy sample is taken from the raised edge of an active lesion where parasites are present. Samples from the necrotic center are avoided. Additional tissue can be obtained through saline aspiration, tissue scrapings, or slit incisions.

For mucocutaneous leishmaniasis, tissue can be obtained through dental scrapings or mucosal granuloma biopsy, although parasites may be difficult to isolate.

For visceral leishmaniasis, the safest way to obtain tissue is through bone-marrow aspiration, although splenic aspiration may be used in cases that are difficult to

diagnose. Splenic aspiration has a higher sensitivity but should be attempted only by experienced physicians.

Treatment

Therapies available in the United States are limited. The 2 available preparations are the pentavalent antimony compounds, sodium stibogluconate (Pentostam), produced in Great Britain, and meglumine antimonate (Glucantime), produced in France. Both have similar efficacy. Depending on the species and region, cure rates of 80-100% have generally been reported.

Sodium stibogluconate is available from the CDC as an investigational drug. Military personnel may receive sodium stibogluconate from the WalterReedArmyMedicalCenter. The efficacy of a 10-day course, with a significantly reduced side-effect profile over the standard 20-day course has been proven.

Amphotericin B is effective against pentavalent antimony-resistant mucocutaneous disease and visceral leishmaniasis. Its use is limited because of its toxic adverse effect profile. The newer lipid preparations are better tolerated and are being used as first-line therapy against visceral leishmaniasis, but the response with cutaneous disease has been mixed. AmBisome is the only US Food and Drug Administration (FDA)–approved drug in the United States available for the treatment of visceral leishmaniasis. AmBisome is also useful to treat antimonial-resistant visceral disease. Single-dose treatment with AmBisome has shown a 91% cure rate in India but is still considered too expensive for general treatment.

Intramuscular pentamidine is effective against visceral leishmaniasis but is associated with persistent diabetes mellitus and disease recurrence.

Because Leishmania species are temperature-sensitive, local treatment with heat or cold provides an alternative to pharmaceutical therapy in some cases. Cryotherapy can be used on small, uncomplicated Old World lesions. A 15- to 20-second freeze-thaw-refreeze cycle over 1-2 weeks was sufficient to cure most uncomplicated cases.

In 2003, the FDA approved the ThermoMed device (ThermoSurgery Technologies, Inc) for the treatment of cutaneous leishmaniasis. This device heats the skin through radiofrequency waves directed to a specified area and depth. A recent study demonstrated a cure rate of 69% at 100 days after treatment. Although the lesions treated in this study were small, the initial results look promising. Further studies may demonstrate this to be a useful therapy for mild disease.

Surgical removal is not recommended for cutaneous disease because of the potential for recurrence at the excision site. Surgery may exacerbate quiescent disease.

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