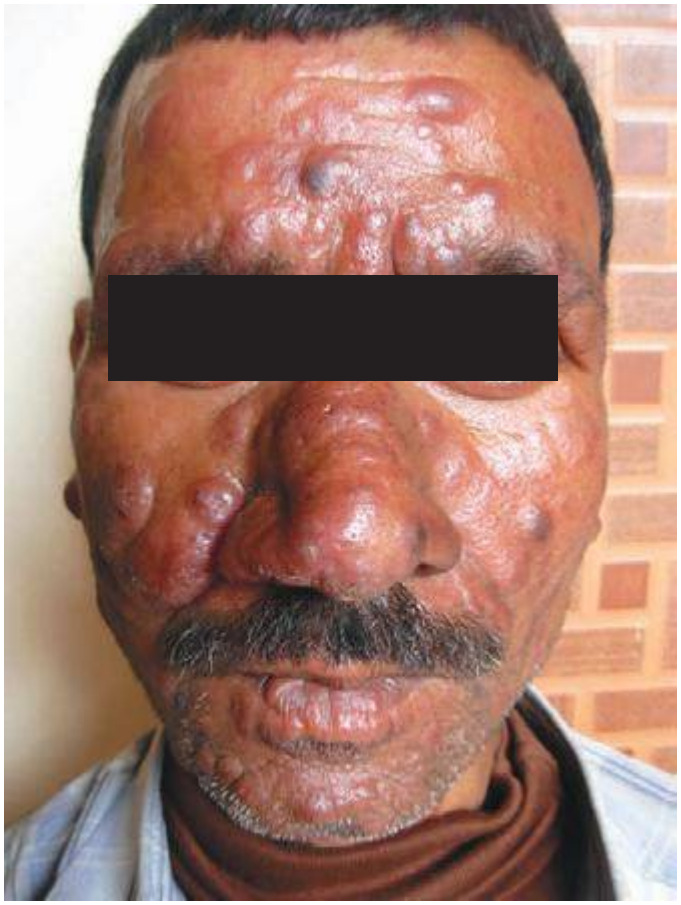

FIGURE 251-4 Post–kala-azar dermal leishmaniasis in an Indian patient. Note nodules of varying size involving the entire face. The face is erythematous, and the surface of some of the large nodules is discolored.

cells. In about half of cases, epithelioid cells—scattered individually or forming compact granulomas—are seen. The diagnosis is based on history and clinical findings, but rK39 and other serologic tests are positive in most cases. Indian PKDL is treated with pentavalent antimonials for 60–120 days. This prolonged course frequently leads to noncompliance. The alternative—several courses of AmB spread over several months—is expensive and unacceptable for most patients. Oral miltefosine for 12 weeks, in the usual daily doses, cures most patients with Indian PKDL. In East Africa, a majority of patients experience spontaneous healing. In those with persistent lesions, the response to 60 days of treatment with a pentavalent antimonial is good.

CUTANEOUS LEISHMANIASIS

CL can be broadly divided into Old World and New World forms. Old World CL caused by *Leishmania tropica* is anthroponotic and is confined to urban or suburban areas throughout its range. Zoonotic CL is most commonly due to *Leishmania major*, which naturally parasitizes several species of desert rodents that act as reservoirs over wide areas of the Middle East, Africa, and central Asia. Local outbreaks of human disease are common. Major outbreaks currently affect Afghanistan and Pakistan in association with refugees and population movement. CL is increasingly seen in tourists and military personnel on mission in CL-endemic regions of countries like Afghanistan and as a co-infection in HIV-infected patients. *Leishmania aethiopica* is restricted to the highlands of Ethiopia, Kenya, and Uganda, where it is a natural parasite of hyraxes. New World CL is mainly zoonotic and is most often caused by *Leishmania mexicana*, *Leishmania (Viannia) panamensis*, and *Leishmania amazonensis*. A wide range of forest animals act as reservoirs, and human infections with these species are predominantly rural. As a result of extensive urbanization and deforestation, *Leishmania (Viannia) braziliensis* has adapted to peridomestic and urban animals, and CL due to this organism is increasingly becoming an urban disease. In the United States, a few cases of CL have been acquired indigenously in Texas.

Immunopathogenesis As in VL, the proinflammatory (T_H1) response in CL may result in either asymptomatic or subclinical infection. However, in some individuals, the immune response causes ulcerative skin lesions, the majority of which heal spontaneously, leaving a scar. Healing is usually followed by immunity to reinfection with that species of parasite.

Clinical Features A few days or weeks after the bite of a sandfly, a papule develops and grows into a nodule that ulcerates over some weeks or months. The base of the ulcer, which is usually painless, consists of necrotic tissue and crusted serum, but secondary bacterial infection sometimes occurs. The margins of the ulcer are raised and indurated. Lesions may be single or multiple and vary in size from 0.5 to >3 cm **(Fig. 251-5)**. Lymphatic spread and lymph gland involvement may be palpable and may precede the appearance of the skin lesion. There may be satellite lesions, especially in *L. major* and *L. tropica* infections. The lesions usually heal spontaneously after 2–15 months. Lesions due to *L. major* and *L. mexicana* tend to heal rapidly, whereas those due to *L. tropica* and parasites of subspecies *Viannia* heal more slowly. In CL caused by *L. tropica*, new lesions—usually scaly, erythematous papules and nodules—develop in the center or periphery of a healed sore, a condition known as *leishmaniasis recidivans*. Lesions of *L. mexicana* and *Leishmania (Viannia) peruviana* closely resemble those seen in the Old World; however, lesions on the pinna of the ear are common, chronic, and destructive in the former infections. *L. mexicana* is responsible for chiclero's ulcer, the so-called self-healing sore of Mexico. CL lesions on exposed body parts (e.g., the face and hands), permanent scar formation, and social stigmatization may cause anxiety and depression and may affect the quality of life of CL patients.

Differential Diagnosis A typical history (an insect bite followed by the events leading to ulceration) in a resident of or a traveler to an endemic focus strongly suggests CL. Cutaneous tuberculosis, fungal infections, leprosy, sarcoidosis, and malignant ulcers are sometimes mistaken for CL.

Laboratory Diagnosis Demonstration of amastigotes in material obtained from a lesion remains the diagnostic gold standard. Microscopic examination of slit skin smears, aspirates, or biopsies of the lesion is used for detection of parasites. Culture of smear or biopsy material may yield *Leishmania*. PCR is more sensitive than microscopy and culture and allows identification of *Leishmania* to the species level. This information is important in decisions about therapy because responses to treatment can vary with the species. Isoenzyme profiling is used to determine species for research purposes.

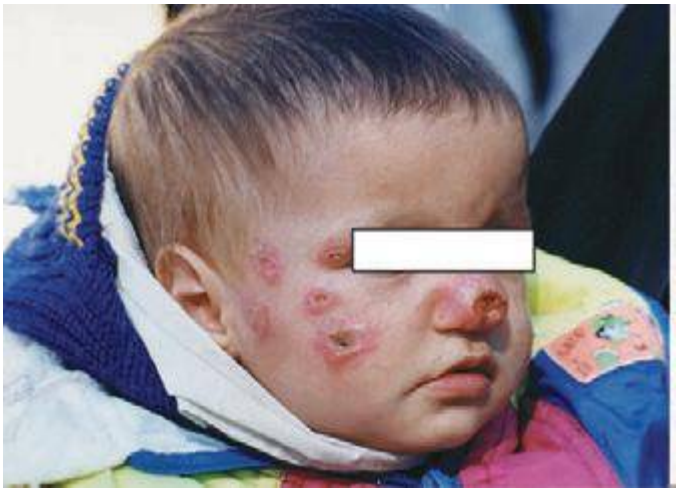

FIGURE 251-5 Cutaneous leishmaniasis in a Bolivian child. There are multiple ulcers resulting from several sandfly bites. The edges of the ulcers are raised. *(Courtesy of P. Desjeux, Retired Medical Officer, World Health Organization, Geneva, Switzerland.)*