Leprosy in Six Isolated Residents of Northern Louisiana

Time-Clustered Cases in an Essentially Nonendemic Area

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• **Northern Louisiana has been essentially free of indigenous leprosy, and now it is not. Six new cases of leprosy have been diagnosed: three in 1986, the other three in 1985, 1983, and 1982, respectively. The patients had been lifelong residents of six scattered rural parishes. Leprosy had never been reported from five of them. No patient had had contact with human leprosy. The patients were white; four were women; the mean ± SD age at onset was 60.3 ± 16.4 years (age range, 31 to 80 years); and the mean ± SD interval to diagnosis was 1.2 ± 1.4 years. One patient had Hodgkin's disease at the age of 25 years and leprosy at the age of 31 years; another patient had cervical carcinoma. All rural northern Louisiana residents coexist with armadillos (*Dasypus novemcinctus*), some of which are infected with *Mycobacterium leprae*, the significance of which is unknown. Hypothetically, exposure to an unknown human case, reactivation of "asymptomatic" leprosy through immunosenescence or immunosuppression, or infection from an environmental source might have occurred. Because the patients lacked contact, travel, residence, and exposure risk factors, the origin of leprosy in the new indigenous cases is noteworthy and is not understood.**

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Leprosy is rare in persons born in the United States and is virtually nonexistent in those who have never lived in an area endemic for leprosy.[1,2] Twenty to 37 cases of endemic leprosy are reported per year in the United States. They account for about 10% of the newly reported cases. Endemic leprosy occurs in persons living in Hawaii, California, Texas, and southern Louisiana.[2,3] Northern Louisiana is not known as a place where transmission of leprosy occurs or for indigenous cases of leprosy.[4,5] It is generally thought that transmission of leprosy requires contact with a case of human leprosy or travel or residence in a region endemic for leprosy, which is presumed to allow transmission from unrecognized cases. However, 50% to 70% of cases of leprosy do not have an identifiable contact with a human being with leprosy, making other explanations plausible.[1,6] Exposure to armadillos, which are common in the south central region of the United States, has recently been shown to be greater in patients with lepromatous leprosy than in controls.[7] We report six recent cases of leprosy in lifelong or nearly lifelong residents of northern Louisiana.

PATIENTS AND METHODS

We investigated six patients recently diagnosed by us or who were referred to us with leprosy. We performed a complete medical history and physical examination; reviewed medical records; obtained and reviewed laboratory tests, x-ray films, and biopsy specimens; conducted visits to each patient's home, sometimes on several occasions; and interviewed patients and family members, particularly about risk factors for leprosy.

We determined the number of cases of leprosy from each parish or county by a review of state health department records for Louisiana, Mississippi, and Texas and of available records from the Gillis W. Long Hansen's Disease Center at Carville, La, by conversations with patients, physicians, and others, and by a literature review.

REPORT OF CASES

Clinical and Demographic Features

The widely scattered residences of the six patients with leprosy are shown on the map of northern Louisiana (Figure). The clinical and demographic features of the patients with leprosy are presented in Table 1. Risk factors for leprosy as related to each of the six cases are summarized in Table 2. Additional clinical details and risk factor analysis are provided for each case in the "Appendix" section.

Common Characteristics

Each of the six patients with leprosy was actually or virtually a lifelong resident of the same immediate area in six parishes in northern Louisiana. Two patients never moved from their birth-

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place. Five parishes had never before reported leprosy in any resident. The cases were separated by at least 30 km (Figure). All of the patients were married or widowed, and white. Four were women. The mean ± SD age at clinical onset was 60.3 ± 16.4 years (age range, 31 to 80 years); the mean ± SD interval to diagnosis was 1.2 ± 1.4 years; and the mean ± SD age at diagnosis was 61.5 ± 17.1 years. In the analysis of risk factors for leprosy (Table 2), the patients lacked contact with any known case of human leprosy, and travel or residence in an endemic region, one of which is usually considered a prerequisite for acquiring leprosy. None of the patients was at risk for, or had antibodies to, human immunodeficiency virus (HIV). All six patients had had indirect contact, and four admitted to direct contact with armadillos, which have been a listed risk factor on the reporting form for leprosy provided by the Centers for Disease Control, Atlanta, since 1976. Armadillos have been found throughout the region, rooting or burrowing in yards and fields or dead and decomposing at the roadside. Therefore, the direct and indirect armadillo contact that we report was not unique to the patients, but was rather a common and inevitable experience for most rural residents in this region.

COMMENT

As we saw these six new cases of leprosy from this essentially nonendemic part of Louisiana during a short period, we determined that while they were separated from each other, they appeared to constitute a regional, time-related cluster of new cases. Because *Mycobacterium leprae* has never been cultivated in vitro, innumerable unanswered questions exist about leprosy. The clinical and epidemiologic cluster of leprosy cases that we describe is consistent with hypotheses that emphasize different aspects of transmission and pathogenesis. Therefore, we explored several, nonexclusive explanations for their recent diagnosis.

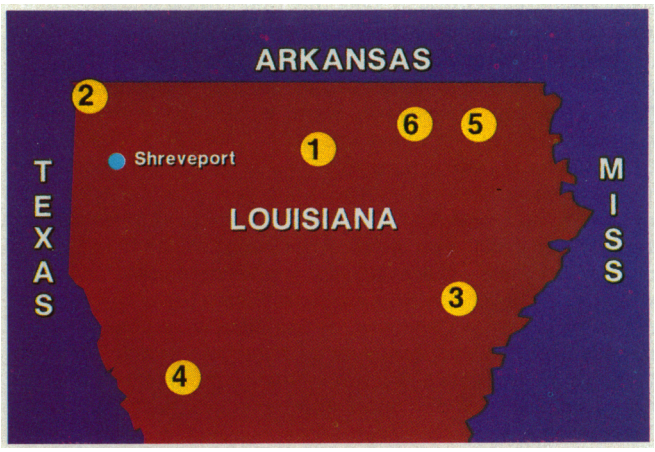
Aging

In some populations where leprosy is decreasing, new cases occur in a more and more aged population. New cases in children and younger adults decrease disproportionately to new cases in aged persons, as leprosy decreases and the area loses its highly endemic character.[1,9,10] That leprosy is a complication of aging is in keeping with our observation that the mean ± SD age at diagnosis was 61.5 ± 17.1 years in our cases. However, it is not logical to assert that our cases mean that leprosy is decreasing in this population, because the cluster of isolated cases that we report represents an absolute increase in regional incidence from zero in lifelong residents. Thus, this cluster of leprosy is not consistent with a decreasing incidence.

Immunosenescence, the immunosuppression that accompanies aging, could make leprosy a complication of aging, and might pertain to five of the cases. For this to have occurred, one must postulate the existence of asymptomatic leprosy or alternatively an increased susceptibility late in life following exposure to a human case or an environmental source of leprosy.

Case Contact

The traditional concept of leprosy transmission is that it occurs only after close, prolonged human contact. The contact is thought to involve nasal secretions. Skin contacts cannot be ruled out. In lepromatous leprosy, nasal mucus contains millions of bacilli that are aerosolized. It is thought that when inhaled, these acid-fast bacilli lead to infection in susceptible persons.[6] Such a hypothetical explanation for our cases requires that exposure to an unknown human case occurred and that our patients, constituting susceptible persons, contracted leprosy. However, detailed interviews, concerning family members, contacts, former owners of the family home, obscure illnesses in household or other contacts, and travel, were elicited from each patient and family. Clearly, no known leprosy case contact existed for any of them. The possibility of six unknown contacts, ie, six living human beings with untreated active, probably lepromatous, leprosy, each of whom was in regular close proximity to one of the six patients, appears to have been highly unlikely.



Map of northern Louisiana showing residence location of each of six sporadic cases of leprosy. MISS indicates Mississippi.

Table 1.—Clinical Features of Six Cases With Leprosy in Northern Louisiana*

	Leprosy							
	Onset		Diagnosis					
Case/Sex	Age, y	Year	Age, y	Year	Clinical Presentation	Leprosy Type†	Underlying Condition	Louisiana Parish
1/F	67	1980	70	1983	Red macules	BL	None	Lincoln
2/F	54	1985	54	1985	Edema, fever	LL	Cervical carcinoma	Rural Caddo
3/F	31	1985	31	1986	Red papules	BL	Hodgkin's disease	Catahoula
4/M	65	1986	65	1986	Red macule	BT	None	Sabine
5/M	79	1986	80	1986	Red macules	LL	None	West Carroll
6/F	66	1979	69	1982	Arthritis	LL	Diabetes mellitus	Morehouse

*See "Appendix" section for details.
†Leprosy type refers to the Ridley-Jopling[8] classification, where LL means lepromatous leprosy; BL, borderline lepromatous leprosy; and BT, borderline tuberculoid leprosy.

Table 2.—Risk Factors for Leprosy in Six Cases in Northern Louisiana*

Case	Family Member, Friend, or Acquaintance With Leprosy	Travel out of US or to Endemic Areas in US	Residences in Addition to Those Cited in Table 1	Leprosy Reported From Home Parish	Armadillo Contact†	
					Direct	Indirect
1	No	No	None	No	No	Yes
2	No	No	Arkansas, 1943-1949, 1951	Yes, see Table 3	Yes, food	Yes
3	No	No	None	No	Yes, food	Yes
4	No	±*‡	Yes*‡	No	Yes, touch	Yes
5	No	No	None	No	Yes, touch	Yes
6	No	No	Simpson Court, Miss before 1956	No	No	Yes

*See "Appendix" section for details. US indicates United States.

†Food means that armadillo meat was prepared in the women's kitchens to eat; neither admitted eating it; touch, that armadillos were handled alive or dead; and indirect, walking barefoot where armadillos rooted and were killed, having armadillos live beneath one's house, drinking water that was potentially contaminated with armadillo excrement, or other means.

‡Case 4's risk of contact with leprosy cases appeared to be nil.

Similarly, it seems untenable, in attempting to explain the average age of the cases, that simply later in life than in a highly endemic area they had had contact with an active case and contracted leprosy.

Asymptomatic or Subclinical Leprosy

The theory of pathogenesis, favoring the existence of an asymptomatic or subclinical leprosy, is based on limited data.[11] The most generalized hypothesis is that nearly everyone gets and carries a subclinical leprosy infection.[12] The idea of asymptomatic leprosy is consistent with widespread but asymptomatic histoplasmosis or tuberculosis in endemic areas. If such a state were to exist, then activation or reactivation of asymptomatic leprosy through immunosenescence of immunosuppression could occur, and might explain isolated cases like these.

If there were an asymptomatic state, immunosenescence could make leprosy a complication of aging. Asymptomatic leprosy is one way to account for the epidemiologic observation that in some populations, new cases are observed mostly in older persons.[1,9,10] The immune systems of elderly persons might permit expression of leprosy, like reactivation tuberculosis that now has its highest incidence in aged persons. The mean ± SD age at diagnosis of 61.5 ± 17.1 years found in our cases is consistent with this possibility. The idea that leprosy is a complication of aging is in keeping with our observations.

Distinguishing prolonged incubation from asymptomatic leprosy is not easy. Recently, the *M leprae*–specific phenolic glycolipid-1 (PGL-1) antigen was detected in blood from a contact of a lepromatous leprosy case two years before the contact developed clinical leprosy.

Immunosuppression and Host Factors

Similarly, if there were an asymptomatic state, immunosuppression could cause leprosy to become clinically apparent. Asymptomatic leprosy or not, immunosuppression might increase susceptibility in persons who are exposed to a human case or to an environmental source, causing them to become ill with leprosy either earlier or in greater numbers than immunologically normal persons. In our patients, an exception to leprosy in aged persons was patient 3 ("Appendix" section); at the age of 31 years, she had already been treated for Hodgkin's disease for 5.5 years. Her case was like two other cases of leprosy, which complicated lymphoma four to five years later.[13] Furthermore, the simultaneous presentation in our case 2 ("Appendix" section) of advanced cervical carcinoma and leprosy is remarkable, suggesting a relationship between the two; however, an association between leprosy and solid tumors has not been made. The potential for persons with HIV infection or with acquired immunodeficiency syndrome to develop opportunistic leprosy should be carefully watched, but only one case has been reported so far.[14] None of our patients was at risk for or had antibodies to HIV. Generally, leprosy does not clinically complicate the usual states of immune compromise, but perhaps this is a misconception, since until recently few severely compromised persons lived long enough to develop leprosy. Leprosy could, therefore, be a complication of immunosuppression, with aging and treated lymphoma being forms of immunosuppression that are supported by our observations.

More broadly, susceptibility to leprosy includes all host factors, from nutrition to genes, even if all are mediated through immune systems. Because nonrelatives in a household with an active lepromatous case get leprosy at a rate of not greater than 5%, genes influence susceptibility.[15] Contrary to earlier reports,[16] HLA antigens, specifically HLA-DR2 and HLA-DQw1, have been associated with leprosy, an observation that has been extended by meta-analysis to many populations (J.R.T., B.C.W., and J. C. McDonald, MD, unpublished data, 1988).[17,18]

Environmental Nonhuman Sources

These patients stimulated our critical review of nonhuman environmental sources for *M leprae*.[6] Infection from the environment might have occurred, for such sources, whether primary or intermediate, are plausible in explaining the origin of *M leprae* for some human leprosy. It is established that *M leprae* exists in the south central United States in the nine-banded armadillo (*Dasypus novemcinctus*). Since leprosy was induced experimentally in this mammal in 1971,[19] and especially since wild armadillos were discovered to harbor it in 1975,[20] there has been concern that the armadillo could spread leprosy to human beings. Homology has been shown between DNA from *M leprae* from a human case and mycobacteria from a leprous armadillo, thus removing the distinction between the organisms.[21] The disease in the two species has similarities.[22]

Proof that *M leprae* is spread to human beings by infected wild armadillos is lacking,[6] but there are some worrisome observations. In Mexicans with lepromatous leprosy, ex-

posure to armadillos was significantly greater than in controls.[7] Seven cases of leprosy in men who handled armadillos in parts of Texas that were known to have leprous armadillos have been reported, with the strong implication that leprosy was acquired in some unspecified manner from the armadillos that they handled and wrestled.[23-25]

An armadillo with a leprosylike disease was found near the home of patient 1; this home was located in the immediate area where all 27 previously reported northern Louisiana armadillos were trapped for survey (see "Appendix" section). The survey showed that eight (29.6%) of 27 had leprosy.[22,26,27] In another survey, 494 armadillos that were found dead at the roadside in Louisiana were studied, including 13 from northern Louisiana. None of the 13 armadillos and ten (2%) of the 494 were positive for leprosy.[28,29] In other surveys, two (10%) of 20 armadillos from coastal Louisiana were leprous,[30] and one of 96 from Mexico was leprous.[31] Of Louisiana armadillo serum samples collected from 1961 to 1964, 17 (9.3%) of 182 and, of recent serum samples, about 20% were positive for specific IgM antibody to PGL-1 antigen.[32] This demonstrated the existence of leprosy in armadillos before their inoculation with *M leprae* in 1968. Although armadillos were known to be in Louisiana before 1920,[33] information about their migration is scant and the time of acquisition of leprosy is unknown. Environmental concepts of leprosy have been advanced with the discovery of PGL-1 antigen in soil.[6] Earthworms near patients' homes, examined as a possible armadillo-leprosy link, however, did not contain acid-fast bacilli.[34]

The two people who were previously described with lymphoma, complicated by leprosy, live in the same general region as our cases, including patient 3 ("Appendix" section) who had Hodgkin's disease.[13] It is noteworthy that one patient was from the east Texas town of Lufkin and the other was from Victoria County, Texas, where more than 7% of armadillos have leprosy.[21] Furthermore, Victoria County is the home of five of the seven reported cases of leprosy in armadillo handlers.

Throughout parts of its range, the armadillo might be a sentinel animal for *M leprae* in the environment. Although it would be incidental to this biologic role, the armadillo brings *M leprae* in contiguity with rural residents in a manner that was totally uncontemplated 15 years ago.[35] Most people in this region have driven past a dead armadillo, every infected one of which is estimated to release 10^{12} *M leprae*.[28]

Possible transmission of leprosy from infected armadillos to human beings has been downplayed because of the extensive casual contact with armadillos that many Louisianans have and because of the low incidence of leprosy.[36] However, if everyone in a population has contact with armadillos, much of it indirect and unnoticed, then comparing the contact in persons with and without leprosy is not meaningful. Yet, apparently that study did just that, ie, interviewed an age- and sex-matched control subject for each rare patient with leprosy from endemic southern Louisiana and drew conclusions.[36] The far greater rarity of leprosy, coupled with the fact that it is essentially not endemic, makes that approach even more problematic in northern Louisiana than in southern Louisiana.

Conclusion

We have reported six cases of leprosy in virtually lifelong residents of this essentially nonendemic region. The newness of these clinical cases makes history itself the control for our observations. Thus, they are a new, endemic cluster of leprosy. Several unresolved theories of pathogenesis and transmission of leprosy can be supported in nonexclusive ways with the observations presented. The cases are rural, dispersed, aged, or immunosuppressed, and they have direct or indirect contacts with armadillos. Everyone in the region coexists with armadillos, some of which are infected with *M leprae*. This contact may pertain in a currently unknown way to these cases.

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APPENDIX: CLINICAL SUMMARY AND ANALYSIS OF RISK FACTORS FOR SIX CASES OF LEPROSY

CASE 1.—This patient presented for a red macule on her thigh that was noted more than one year earlier. It enlarged, a new one became visible, and both were anesthetic. A skin biopsy specimen showed chronic granulomatous inflammation with numerous acid-fast bacilli, consistent with borderline lepromatous leprosy. From Louisiana State University Hospital, Shreveport, she was referred to the Gillis W. Long Hansen's Disease Center in Carville, La. She received outpatient treatment for three years with dapsone and rifampin, and she has continued treatment with dapsone. She has nearly recovered fully.

This patient had had no risk factor for leprosy, except contact with armadillos. She was born on a farm and had lived there her entire life. Armadillos were ubiquitous there after 1940, and were regarded as a nuisance. They burrowed by the shallow well and under her house, which had floorboards between which dust could enter. Her husband shot many armadillos in their yard, spilling blood and discarding bodies nearby. She often went barefoot where the armadillos rooted and were killed. Near the house was a worm "farm," ie, soil kept moist to grow fishing worms, which attracted armadillos. That dirt got trafficked inside. Hence, various human contacts with armadillos, their blood, and other body fluids occurred.

In December 1985, biopsy specimens were taken from an armadillo that was killed nearby by a vehicle but not mutilated. Ear specimens that were fixed, sectioned, and stained (hematoxylin-eosin) showed no granulomas or inflammation, but a Fite stain revealed numerous acid-fast bacilli singly and in clusters, typical of armadillo leprosy. Of note is that in the survey of northern Louisiana armadillos, all were trapped in this area, and eight (29.6%) of 27 had leprosy.

CASE 2.—This patient was referred to the Louisiana State University Hospital for cervical carcinoma. In consultation for fever, we found peculiar ankle edema and chronic erythema. A skin biopsy specimen showed foamy macrophages; a Fite stain showed numerous acid-fast bacilli, some within cutaneous nerves. Many erythematous lesions and extensive edema of both legs quickly developed. She was treated with dapsone and rifampin while undergoing cancer therapy. After developing jaundice and erythema nodosum leprosum, she was referred to the Gillis W. Long Hansen's Disease Center, where rifampin was discontinued. Skin scrapings showed numerous acid-fast bacilli, consistent with lepromatous leprosy. She has had a gradual response to dapsone.

Risk factors included her birth in Caddo Parish, Louisiana, the only parish in this report from which leprosy has previously been reported, and armadillo contact. Caddo Parish is urban because of Shreveport; however, she was born and lives in a rural area, 50 km from Shreveport. She lived for six years in Arkansas, a state with no endemic leprosy. Of eight cases reported from Caddo Parish, only one other was from a rural area like case 2 (Table 3); that Texan lived in Caddo Parish after the age of 2 years, except for a year in Wichita Falls, Tex, and several months in San Antonio, Tex (1918), at which time he was healthy, but where he might have been exposed. Lepromatous leprosy was found in 1919, and reported in 1924 from a town that no longer exists. He had had no known leprosy contacts. He had leprosy from 1919 to 1924, while living 16 km from where our patient 2 was born in 1931. This patient, her parents, and friends who were born there in 1899 could not establish any contact or connection with him, using names and places. No other case occurred there. Other cases were imported or appeared to be irrelevant to case 2 and each other. Hence, evidence does not favor human-to-human transmission of leprosy in Caddo Parish.

This patient first saw armadillos as a child. They burrowed under her home and were sometimes destroyed in the yard. They were active as shown by burrows and rooting signs, attracted by worms and a bayou. The spring was surrounded by armadillo burrows. She often went barefoot. A year before presentation, she lacerated her ankle on a stump. The unhealed ankle was scratched by a rooster while she was barefoot in the chicken yard. Armadillos frequented these areas. The wound had not healed when she presented; it was the first site affected by clinical leprosy, and it healed with antileprosy drug treatment.

CASE 3.—This patient observed red papules on her ankle. In a month, edema developed, and the papules became tender, causing her to present to Louisiana State University Hospital in November 1985. She thought they might be Hodgkin's disease, which had been diagnosed in 1980. She had undergone a staging laparotomy

Table 3.—Analysis of Case 2 With Reference to Leprosy in Caddo Parish, Louisiana*

Case/Sex	Birthplace, y†	Place Reported From, y	Year of Death or Follow-up‡	Comment
1/M	Lufkin, Tex, 1907	Caddo City, La, 1924	Died, 1935	See text; long visit to San Antonio, Tex, in 1918
2/M	Caddo Parish	Keithville, La, 1925	Died, 1926	Shreveport area, 55 km from our case 2
3/F	Caddo Parish	Shreveport, La, 1932	Died, 1933	. . .
4/M	Caddo Parish	Shreveport, 1954	Follow-up, 1959, NEAL	Manila, Philippines; Korea; Hawaii in World War II
5/F	India, 1942	Shreveport, 1959	Follow-up, 1979, NEAL	Father had leprosy
6/F	Louisiana, 1934	Shreveport, 1982	Follow-up, 1985, NEAL	Lived >2 y in Philippines
7/F	Burma, 1933	Shreveport, 1984	Follow-up, 1984, NEAL	Came to US in 1971
8/F	Our case 2, 1931	Vivian, La, 1985	Follow-up, 1988	Dapsone treatment; see text

*Data from the Gillis W. Long Hansen's Disease Center, Carville, La, referring physicians, and local medical records.
†Birthdate unknown for patients 2 through 4.
‡NEAL indicates no evidence of active leprosy.

with splenectomy, responded to chemotherapy, and relapsed in 1982. A second course of chemotherapy caused remission.

Skin biopsy specimens showed granulomatous inflammation and acid-fast bacilli (Fite stain) in extracellular clusters, in macrophages, and inside cutaneous nerves, consistent with lepromatous leprosy. The papules became larger, more numerous, and more tender, and they appeared on her arms and face. Therapy with dapsone and rifampin has led to resolution of the skin lesions.

Never before has leprosy been reported from Catahoula Parish, Louisiana, but a man born there in 1934 was reported to be from St Landry Parish, Louisiana, in 1982. This white man was born 25 km from the home of patient 3, but from 1946 to 1958, he lived in Rapides Parish, Louisiana, and from 1958 in St Landry Parish. Those parishes are known for endemic leprosy and are considered the source of his leprosy. Risk factors appeared to be limited to armadillos. This patient had lived on one rural property. Armadillos lived there in a slough and burrowed near the well. She had lived partly at the home of her parents-in-law. Armadillos lived near it and under it, partly attracted by a fishing bait worm farm. Her father-in-law had killed a "boxcarful" of armadillos; he disposed of them nearby.

CASE 4.—This patient presented to the Louisiana State University Hospital with a red 5-cm papular lesion on his left shoulder, which had begun three months earlier as an erythematous ringlike macule. He had had the onset of a 10-kg weight loss five months earlier, and swelling of his left hand and hyperuricemia one month before admission, which was treated elsewhere as gout. He also had small red papules on his left arm, trunk, and abdomen; erythematous macules numbered ten each on his left arm and chest and six on his right arm. A severe, mainly motor neuropathy in his left arm resulted in loss of muscle mass and was particularly noticeable because he was a carpenter and guitarist. A left brachial arteriogram showed obstruction of the ulnar artery, increased vascularity around the distal ulna and in the hand, and advanced bone erosions in the hand, consistent with inflammation, synovitis, and periarticular vasculitis. Skin biopsy specimens showed granulomatous inflammation, but only the seventh specimen showed three beaded acid-fast bacilli (Fite stain), thus confirming borderline tuberculoid leprosy. To dapsone and rifampin treatment was added prednisone for management of neuropathy. His skin lesions and left arm edema have gradually improved; the neuropathy has not progressed.

This patient was born and lived in rural Sabine Parish, Louisiana, from which leprosy has never before been reported. He also lived in Texas at Anáhuac, Chambers County, from 1952 to 1962. Two men with leprosy were reported from Chambers County. The first had come from Vacherie, La (born in 1911), and lived in Hankamer in Chambers County when his leprosy was diagnosed in 1935; he was treated at Carville and was unavailable for follow-up in 1960; his brother died of leprosy at Carville. The second was a Mexican laborer (born in 1941) whose leprosy was diagnosed in 1976. Both contracted it elsewhere, and the one antedating the patient (case 4) did not live in Anáhuac. Case 4 also lived at Fort Hood, Bell County, Tex, from 1941 to 1946, which also appeared to be irrelevant because the first case from Bell County was in 1973.

As a carpenter who worked outdoors near his home, he was well aware of having indirect contact with armadillos in his rural environment. Some lived near his home, burrowing near his shallow water well.

CASE 5.—This patient was an active man who had onset of splotchy red macules on his trunk in April 1986. Within two months, he had nodular red lesions on his torso and macular and papular red skin lesions on all extremities. His internist suspected leprosy. A skin biopsy specimen showed inflammation in the dermis, perineural inflammation, and macrophages with large numbers of acid-fast bacilli (Fite stain). The diagnosis of lepromatous leprosy was classified as subpolar at the Gillis W. Long Hansen's Disease Center. After five months of dapsone and rifampin treatment, erythema nodosum leprosum developed and later resolved.

Patient 5 has lived only in West Carroll Parish, Louisiana. Leprosy had never been reported from this rural parish before, although a previously described patient with leprosy from the city of Monroe, La (Ouachita Parish), in 1980 had been born in West Carroll Parish, Louisiana, in 1947. That white man served in the US Navy and went to ports of call in leprosy-endemic countries. Patient 5 first saw an armadillo in 1914. It was alive and displayed as a novelty by local hunters. In both sport and employment this patient was outdoors, so he had had direct and indirect contact with armadillos since they became common in the 1930s. This included killing two to three armadillos weekly near his homes and disposing of the carcasses by hand.

CASE 6.—This elderly diabetic patient had had the onset, poorly remembered, of a discolored lesion on a wrist, for which she saw a physician in 1979. Arthritis was diagnosed, for which she received steroid and other therapy. From 1979 to 1981, the rash extended. In 1982, a dermatologist found an erythematous maculopapular dermatitis of her face and extremities and diagnosed lepromatous leprosy with skin biopsy specimens. The Gillis W. Long Hansen's Disease Center confirmed it, and therapy with dapsone and rifampin was begun. She discontinued taking rifampin after 18 months; many purple nodules developed that suggested Kaposi's sarcoma to dermatologists, but she had lepromatous leprosy. Poor compliance contributed to her relapse. Since 1986, she has responded to the addition of clofazimine to dapsone treatment.

The risk factors in patient 6 were limited. She lived in rural Simpson County, Mississippi, before moving to a town in Morehouse Parish, Louisiana, in 1956. Before her, no one with leprosy has been known to have been born in or reported to be from Simpson County or Morehouse Parish. She had definite indirect contact with armadillos while hunting and fishing with her husband from 1956 until his death in 1974. She had had no contact with armadillos at her home or yard, having lived in an older frame house in town for 32 years.