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THE LUCIO FORM OF DIFFUSE LEPROSY CASE IN A LOUISIANA NEGRO ¹

VINCENT J. DERBES, M.D., MONROE SAMUELS, M.D.,
OLLIE P. WILLIAMS, M.D. AND JOHN J. WALSH, M.D.²

New Orleans, Louisiana

It was in 1852 that Lucio and Alvarado described a form of leprosy as "Lepra manchada ó lazarina" which had been observed by them at the San Lázaro Hospital in Mexico City. Although their description was excellent, this form of the disease was essentially forgotten until it was restudied by Prof. Fernando Latapí. In addition to circumscribed, erythematous spots ("manchas"), chiefly on the extremities, which went on to vesiculation, superficial necrosis and scarring, other salient features were summarized by Lucio and Alvarado (p. 10) as follows: "In general, the loss of the eyebrows is of such value for diagnosis, that united with the diminution of sensitivity and the disease of the nasal mucosa which we will mention later, without there being any other alteration, one may be sure that an individual is attacked by the disease of San Lázaro, and that this will very probably manifest itself in the spotted ('Manchada') form." Lucio also stressed the poor prognosis.

In a series of papers Latapí and other Mexican dermatologists elaborated further details. In a lecture given before the Pacific Dermatologic Association which met in Mexico City in 1955 Latapí referred to: "The diffuse generalized infiltration of the skin, at the beginning 'succulent,' especially on the face and hands, giving a healthy appearance (which someone whose name we do not remember called 'lepra bonita,' pretty leprosy) but also reaching the stage of moon face, and later giving rise to atrophy, as in senile skin. On the limbs, particularly the lower ones, generalized infiltration with innumerable terminal neuritis causing anhidrosis, dryness and even a frankly ichthyosiform aspect." Arnold has pointed out that in many respects diffuse leprosy

¹ Reprinted, by permission, from an article entitled "Diffuse Leprosy" in the *Archives of Dermatology* **81** (1960) 210-224, with copies of the original illustrations supplied by the senior author.

² From the Department of Medicine, Tulane University School of Medicine and the Charity Hospital of Louisiana (Drs. Derbes and Williams); the Department of Pathology, Louisiana State University School of Medicine and the Charity Hospital of Louisiana (Dr. Samuels); and the U.S.P.H.S. Hospital (Dr. Walsh, senior surgeon, U.S.P.H.S.).

is like ordinary lepromatous leprosy: the gradual onset, the generalized involvement of the skin, the relatively symmetrical nerve involvement, the nasal lesions, the loss of eyebrows, the abundant bacilli, the lepromatous histology, the frequent positive tests for syphilis, the negative Fernandez (48-hour) and Mitsuda (3-week) lepromin tests and the progressive downhill course. In other respects, Arnold continues, it is unlike lepromatous leprosy: lesions of the eye and larynx are rare, the Medina (4-5 hour) lepromin test is often positive, and most extraordinary of all, the classic and almost definitive lesions of lepromatous leprosy, the circumscribed granuloma, the leproma itself, never occurs.

It is now clear that Lucio was in error in believing that this aspect of the disease is confined to Mexico. Thus, Romero estimates that 30% of the cases in Costa Rica are of this type. Obermayer reported a case from California, and Arnold one from Hawaii. It has been stated to occur also in Brasil, Argentina, and Spain (the last questioned by Dr. Félix Contreras of Madrid). Regardless of the ultimate distribution of the Lucio phenomenon, it seems safe to accept as verified Latapi's prediction that it will be found in other places as soon as it becomes better known.

In the case presented herewith, the patient has never left his native state of Louisiana. Obermayer's patient was a native born American of Mexican parents who may well have contracted his disease in California, but who did visit Sonora, where his parents were born, for six months. In view of the failure both of Obermayer's patient and of our own to receive a correct diagnosis promptly, in spite of being seen by experienced dermatologists, it was considered desirable to report this case.

REPORT OF A CASE

The patient, admitted to Charity Hospital Nov. 5, 1956, is a 52-year-old Negro sawmill worker, and a life-long resident of Alexandria, La. He has never been out of this state, even for short visits. In his view, he was perfectly well until 1951, when he was struck on both legs by a saw. The injured sites became "infected," extended, and rendered him unable to walk. Over the next few years he developed new lesions, not only of the legs and feet, but also of the arms and hands. Initially, there is a small purple hyperpigmented macule, which becomes vesiculated, then encrusted and ulcerated. It heals leaving a soft depigmented scar with almost geometrical contours; some of these scars are triangular, some parallelograms, and some polyhedral. He was treated by a number of physicians and then admitted to a regional veterans hospital on two occasions, in 1955 and 1956. He was considered by the then consulting dermatologist to have pyoderma and syphilis, the latter diagnosis based on positive serologic tests. After treatment with penicillin these tests reverted to normal, but there was little or no improvement in the skin lesions.

He has had frequent episodes of epistaxis for the past several years. There has been a loss of 47 pounds since onset of illness (160 to 113). There has been recurrent, moderate pain in the legs. He lives with aged parents, but neither they, nor any of his friends, have similar disease.

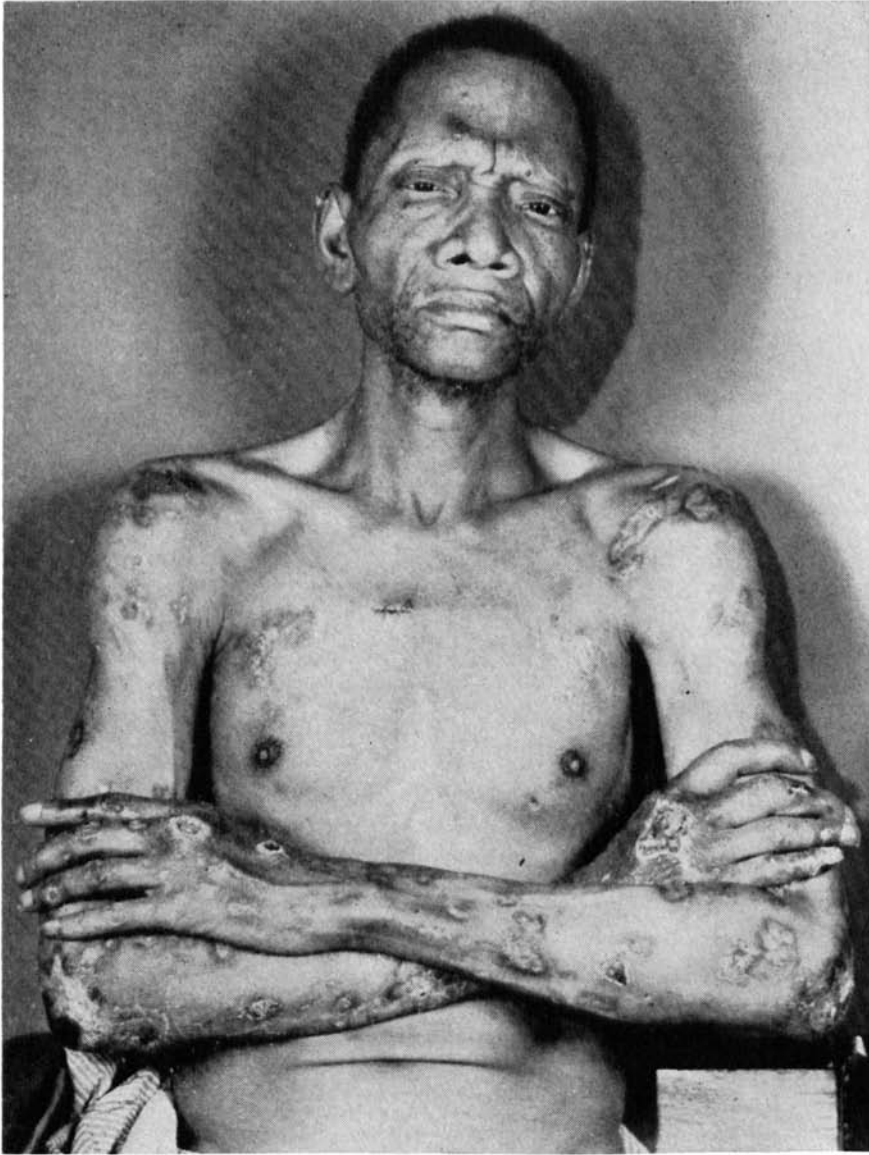


FIG. 1. Clinical appearance of patient. To be noted particularly are the polygonal scars, ulcers over the knuckles and elbows, lack of lepromata, and absence of eyebrows and eyelashes.

On physical examination one sees a thin, emaciated, chronically-ill person in no acute distress (Fig. 1). Vital signs: temperature 97.8, pulse rate 128 per minute, respiratory rate 20 per minute, blood pressure 100/70. There is severe atrophic rhinitis with crusting and frank pus in the nose. There is bilateral sinusitis, as well as crusting and pustulation of the external ears. There is atrophic pharyngitis with slight hyperpigmentation of the mucosa of the throat. Poor oral hygiene and numerous carious teeth are seen. The remainder of the general examination is within normal limits except that there is clear-cut atrophy of the muscles of the lower extremities. He is unable to walk.

The neurological examination (Dr. Ruth Paterson) discloses normal findings with reference to cranial nerves and deep tendon reflexes. There is peripheral neuropathy of the lower extremities and both ulnar nerves are palpable.

There is definite loss of pain sensation over the area served by the ulnar nerves; loss of light touch sensation over the dorsum of the hands and distal dorsal aspect of the forearms, and the distal 3rd and 5th phalanges bilaterally. There is diminished sensation in both feet associated with numbness. There is motor function pain in the hands, and right middle finger drop. There are no contractures. Examination of the eyes, including slit-lamp study, discloses normal findings.

The skin survey discloses complete loss of eyebrows and eyelashes (Fig. 2). There is thinning of the scalp hair generally but especially over the occiput and nape of the neck. Several small (1-2 cm.) hyperpigmented macules are noted on the flexor surfaces of the forearms and on the anterior chest and left cervical region. A distinct erythematous border is seen on each of these lesions.

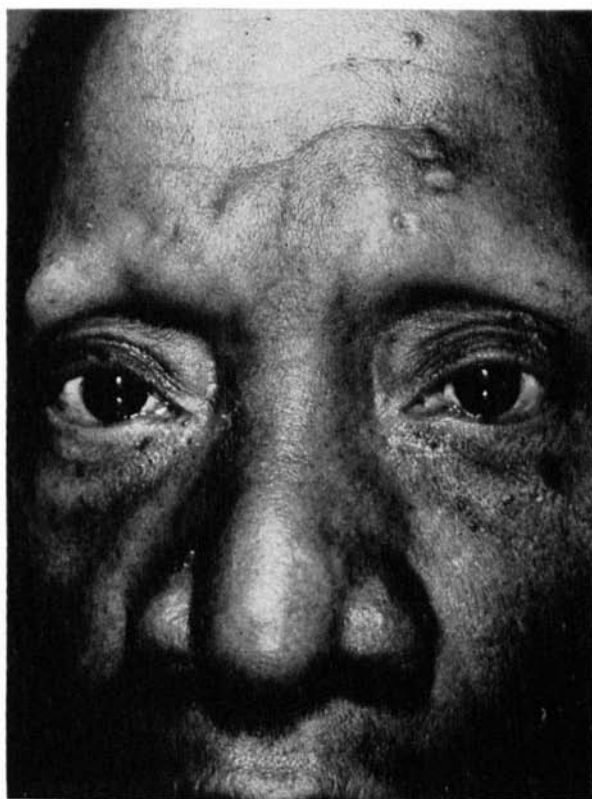


FIG. 2. Close-up view to emphasize the madarosis. Scars over brow represent biopsy sites.

A 5 × 5 cm., deep, ulcerous pustular encrusted lesion is present on the dorsum of the right hand overlying the second and fourth metacarpals and extending very deep into the subcutaneous tissue, apparently involving the tendons. The periphery of the lesion reveals healing with granulomatous reaction.

There are encrusted vesicular plaques 1-3 cm. in diameter on the skin of the dorsal and ventral surface of arms and forearms, chest, neck, upper back, and lower legs.

Numerous depigmented, superficial scarred plaques 1-3 cm. are found on the arms, neck and chest, and lower legs. Several large, encrusted, pustular, granulomatous ulcers are observed about the ankle and lower legs bilaterally, with a very intense stasis dermatitis and hyperpigmentation of the surrounding skin. Some of the areas appear to

be possible early gangrene, but perhaps this represents a hyperpigmentary response to previous inflammation.

No lepromata are present.

The impression on admission was acquired epidermolysis bullosa versus factitial dermatitis. The patient was presented to the Louisiana Dermatology Society and the prevailing opinion was that the lesions were self-induced. No clinician suggested the possibility of leprosy. The diagnosis was established by the pathologists.

Laboratory Data.—Smears from nasal mucosa, ear lobe, and "normal" skin contain enormous amounts of acid-fast bacilli.

BIOPSY SPECIMENS

Skin.—Eyebrow: The epithelium shows some atrophy and a slight degree of hyperkeratosis (Fig. 3). There is plugging of the hair follicles with keratotic material, but the follicles are not atrophic, and hairs are present in the deeper portions of the follicles. Some of the follicles show organisms compatible with *Demodex*. The sebaceous glands are normal in appearance. Scattered throughout the corium, in both the superficial and the deeper portions, are focal collections of fairly large cells with pale-staining foamy cytoplasm. Some of these collections are arranged around some of the smaller blood vessels. No inflammatory infiltrate of an acute or chronic nature is seen around these cells. Acid-fast stain on the tissue shows the foam cells to contain large numbers of acid-fast bacilli (Fig. 4).

Lesion on Arm.—The epithelium is atrophic. There is also some atrophy and keratotic plugging of the hair follicles. The subepithelial collagen is slightly hyalinized and shows a decreased number of nuclei. Scattered small foci of "foam" cells are seen in the upper corium, some of them around blood vessels. These foci are infiltrated with polymorphonuclear leukocytes, lymphocytes, plasma cells, and nonvacuolated macrophages. The most

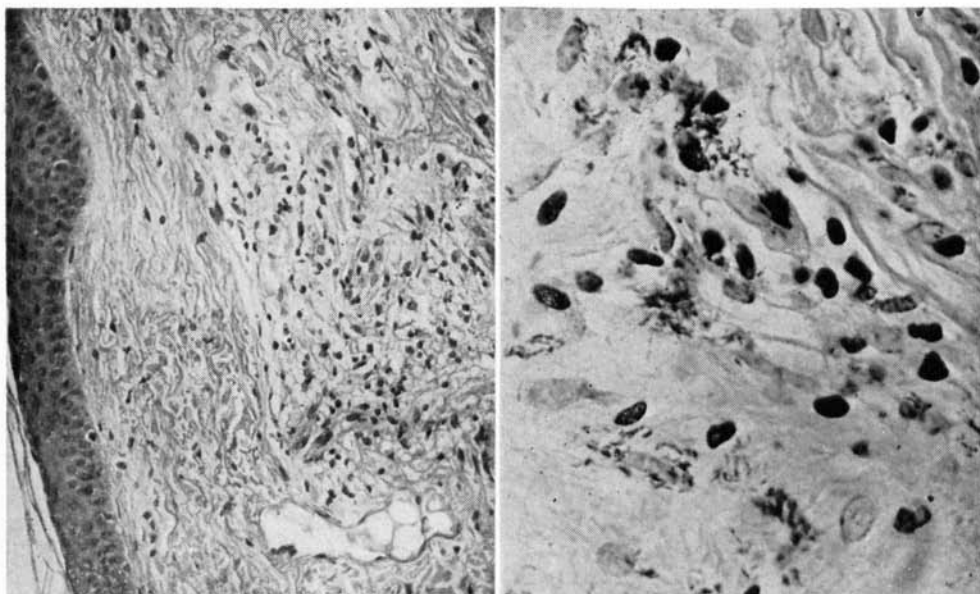


FIG. 3. Section of skin from eyebrow, showing the typical lesion of lepromatous leprosy. There is slight atrophy of the epidermis and large accumulations of foamy "lepra" cells in the corium. Hematoxylin and eosin.

FIG. 4. Acid-fast stain on section 1, showing the large numbers of lepra bacilli. Fite acid-fast stain.

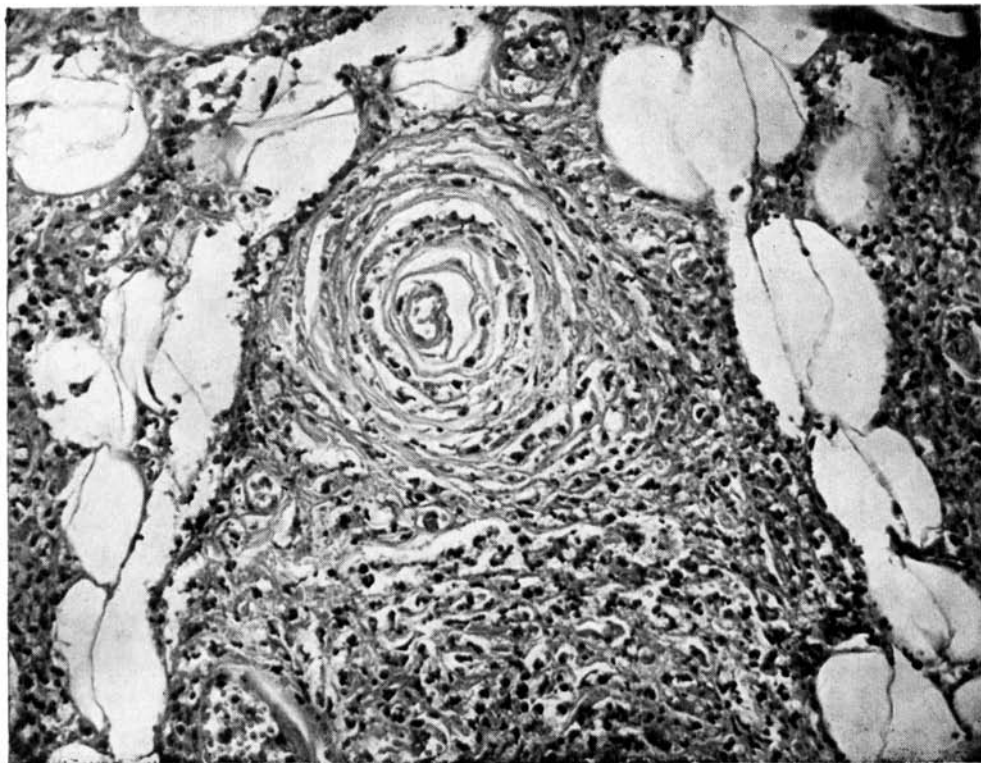


FIG. 5. Section through ulcerated area on arm. Artery showing marked acute arteritis and acute inflammatory infiltrate in the surrounding tissue. Hematoxylin and eosin.

striking lesion is an acute vasculitis and thrombosis of some of the deeper blood vessels (Fig. 5). There is marked thickening of the vessel wall and infiltration with acute and chronic inflammatory cells (Fig. 6). The lumens of some of the vessels contain hyaline thrombi. There are scattered areas of necrosis, particularly about some of the sweat glands and also within the adipose tissue in the deep corium (Fig. 7). The necrotic lesions show infiltration with polymorphonuclear leukocytes, lymphocytes, plasma cells, and occasional foamy macrophages.

Acid-fast stain on the tissue shows large numbers of acid-fast bacilli in the areas of vasculitis (Figs. 6 and 8), in the necrotic areas, and also within the foam cells in the upper corium.

Liver.—There is a periportal infiltration consisting of lymphocytes, macrophages, and rare eosinophils and polymorphonuclear leukocytes. Many of the macrophages have voluminous, foamy cytoplasm (Fig. 9). In addition, there are focal infiltrates of foam cells throughout the parenchyma of the liver, some around the central veins, and others midzonal in location. Many of the Kupfer cells show a similar type of foamy cytoplasm. The parenchymal cells of the "liver are essentially normal in appearance.

Acid-fast stain shows large numbers of acid-fast bacilli within the previously described foam cells (Fig. 10).

Special stains for the presence of amyloid are negative.

Kidney.—The section of the kidney stained with hematoxylin and eosin is essentially normal in appearance.

Special stains for acid-fast bacilli and for amyloid are negative.

Peripheral Blood.—Blood was drawn from the patient and double oxalate used as

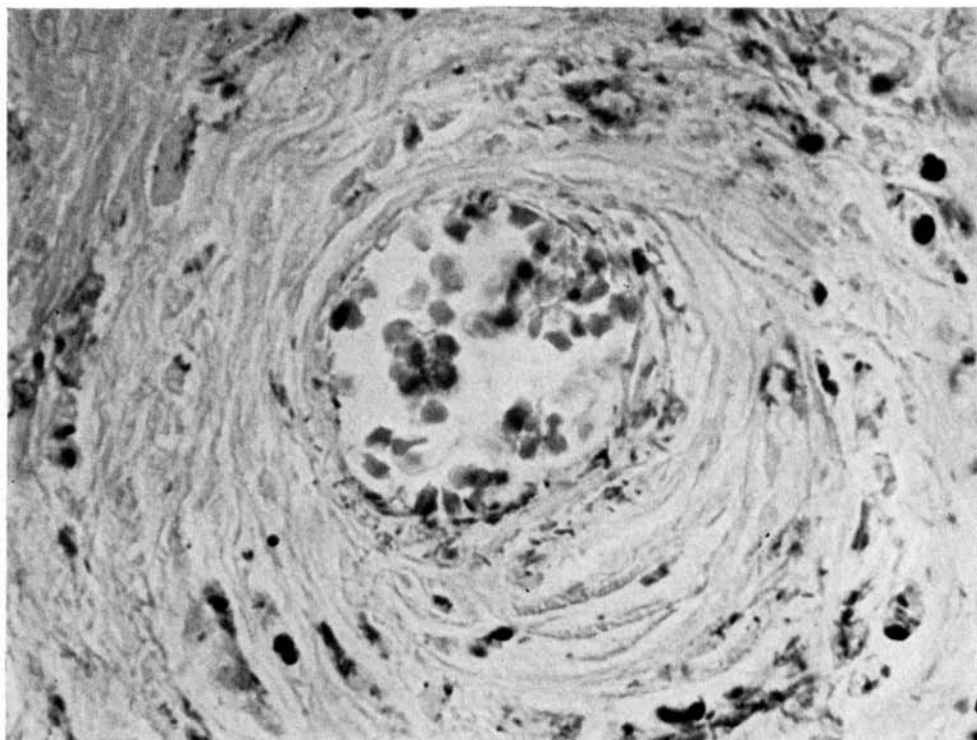


FIG. 6. Section through artery. There are large numbers of acid-fast bacilli in the intima and the wall of the vessel. Fite acid-fast stain.

an anticoagulant. The blood was spun in a Wintrobe hematocrit tube, and the buffy coat of white cells was aspirated and smeared on glass slides. Acid-fast stain on this material showed rare cells filled with acid-fast bacilli to be present in the peripheral blood.

Bone Marrow.—The erythroid, granulocytic, and megakaryocytic elements of the marrow are normal. There are increased numbers of plasma cells seen. The red cells on the smear show marked rouleaux formation. There are scattered macrophages which have a vacuolated, foamy cytoplasm. These range from cells showing but one cytoplasmic vacuole, to cells in which the vacuoles have displaced the nucleus to one side of the cell and have distorted the shape of the nucleus (Fig. 11).

Acid-fast stains on the marrow smears show these vacuolated cells to be filled with acid-fast bacilli (Fig. 12).

There is moderately hypochromic, slightly microcytic, anemia with a moderate number of target cells. Hemoglobin 9.7 gm., 4,140,000 red blood cells per cubic millimeter, packed cell volume 32%, mean corpuscular volume 80, mean corpuscular hemoglobin concentration 30, thrombocytes 386,000 per cubic millimeter, white blood cells 10,500 per cubic millimeter: 81% polymorphonuclear leukocytes, 1% eosinophils, 5% monocytes, 13% lymphocytes. There were 1.6% reticulocytes. The erythrocyte sedimentation rate was: uncorrected 53 mm. per hour, corrected 25 mm. per hour. Clotting time: 7 minutes 30 seconds. Bleeding time: 2 minutes 30 seconds. Prothrombin time: 16.8 seconds. Total serum protein 8.96 gm. %: albumin-globulin ratio 2.86:6.10. Cephalin flocculation: 3+, thymol turbidity 9.2 units. Alkaline phosphatase 15.96, acid phosphatase 1.55, prostatic acid 0.07. Bromsulphthalein retention: 34%. Bilirubin 0.81 mg., calcium 5.0 milliequivalent per liter, phosphorous 2 mEq/l., chloride: 91.6 mEq/l.,

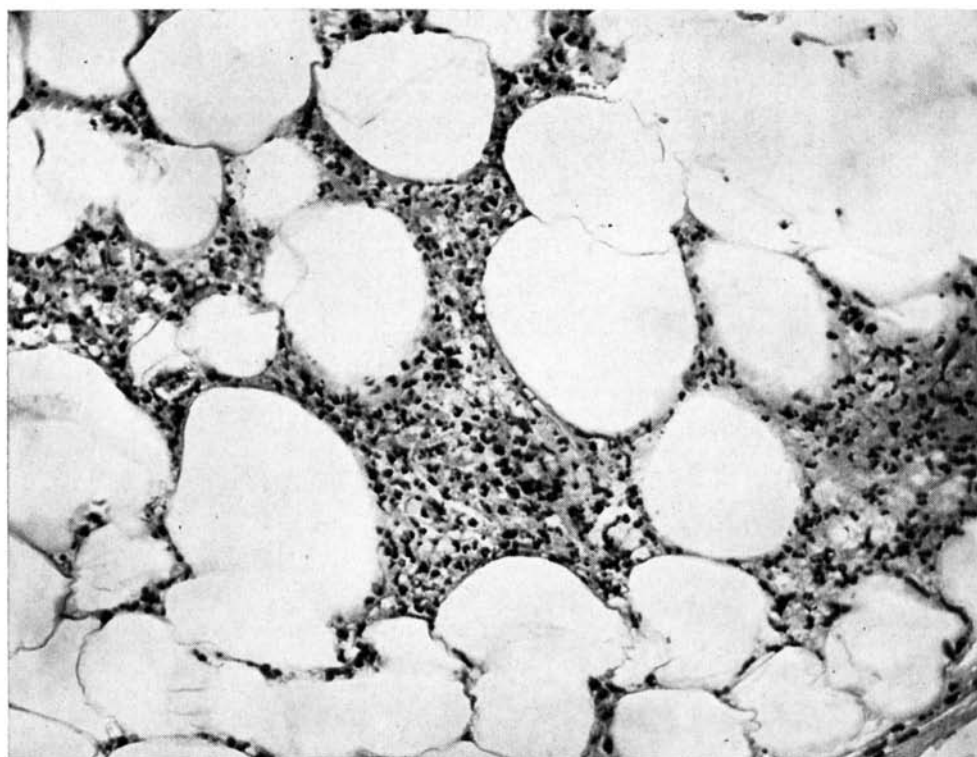


FIG. 7. Section through ulcerated area on arm. There is marked panniculitis with areas of necrosis and acute inflammation extending deep into the subcutaneous fatty tissue. Hematoxylin and eosin. Acid-fast stains showed numerous acid-fast bacilli.

cholesterol 148 mg., blood urea nitrogen 9.0 mg., fasting blood sugar 118 mg. Serology: blood VDRL 16 dils.; Kolmer positive. Spinal: VDRL and Kolmer positive. TPI test for syphilis done at United States Public Health Service Laboratory, Chamblée, Georgia, negative. Total protein 7.48 gm. %, γ -globulin 4.59 gm. (61.3%), albumin 2.89 (38.7%).

Urine pH 5.5, specific gravity 1.014, albumin negative, sugar negative, microscopic negative. Fishberg concentration: 1.020, 1.019, 1.013.

Skin Tests.—

Lepromin: Negative Medina (4 hours), Fernandez (48 hours) Mitsuda (3 weeks).

Tuberculin: Negative 24 and 48 hours.

Blastomycin: Negative 24 and 48 hours.

Histoplasmin: Negative 24 and 48 hours.

Coccidioidin: Negative 24 and 48 hours.

Histamine test: Wheal only, no flare or purplish discoloration.

Congo red skin test: Strongly positive.

X-rays.—

Chest: Old pleural reaction of both costophrenic angles.

G.I. Series: Essentially normal.

Views of spine, skull, long bones, wrists, Water's view of sinuses, KUB, intravenous pyelogram, normal.

Thorne test for adrenal function showed normal 50% drop in eosinophils.

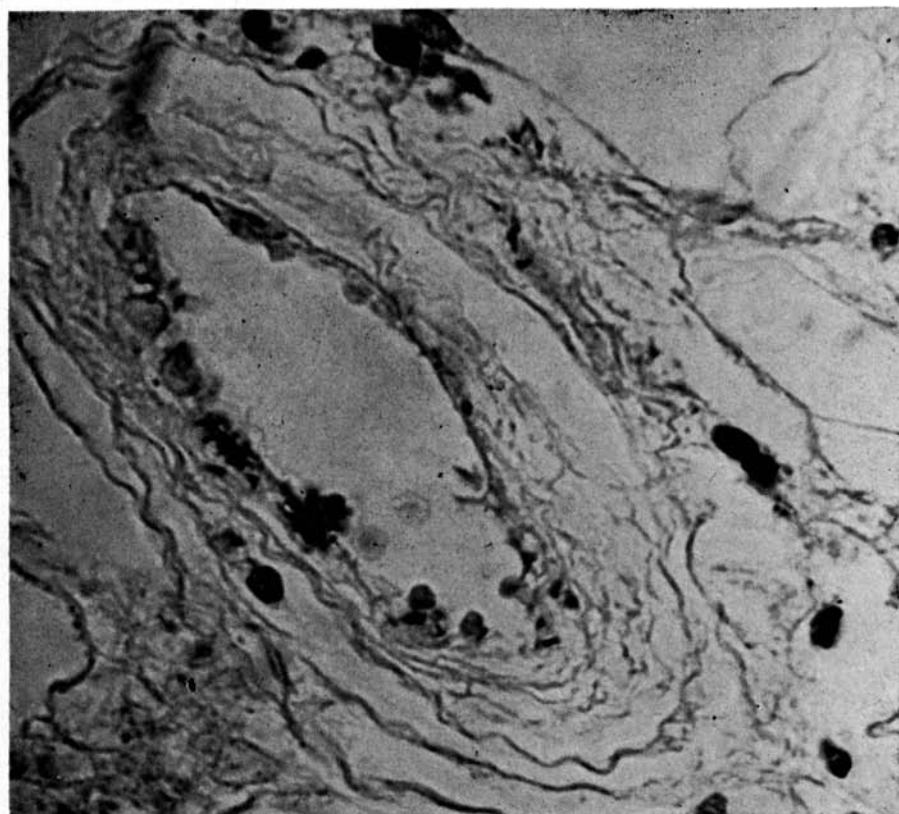


FIG. 8. Vein showing acid-fast bacilli in the intimal cells. Acid-fast stain.

COMMENT

As we are not leprologists we sought the opinions of more learned men. To this end, kodachromes, histologic sections, and the protocol of the case were sent to a number of outstanding persons who had studied this phase of leprosy. It is but natural that we did not hear from all. The comments of those who did favor us are listed below:

HARRY L. ARNOLD JR., M.D., Honolulu, Hawaii:

A review of your most interesting protocol, and the Kodachromes and the histologic specimens, convinces me that you have indeed a case of diffuse lepromatous leprosy with the Lucio phenomenon—plus, or at least so I. L. Tilden and I would think, severe chronic nodular vasculitis with acute phases, possibly on the basis of leprosy. Involvement of these larger vessels is not ordinarily a part of the Lucio phenomenon (though what we know about this is what Latapí has told us, and he would be the best source of an opinion on this score). (Larger vessels may be involved. Latapí.)

Points worth special mention are as follows: (1) absence of eye involvement; in an ordinary lepromatous case one might expect some involvement at this stage, though perhaps it is a little early; (2) relative abundance of neutrophils in the sections—normally absent in lepromatous leprosy; (3) even more striking hyperglobulinemia than usual, with false-positive serologic tests for syphilis, much more regularly found in these diffuse cases than in ordinary lepromatous cases.



FIG. 9. Needle biopsy of liver. There is an infiltrate of "foam cells" around one of the central veins. Hematoxylin and eosin.

The deep ulcerative lesions are unusual, I believe, in Lucio's leprosy—I suppose the involvement of larger vessels is responsible for them.

Your finding of *M. leprae* in bone marrow and circulating leukocytes (or were they circulating histiocytes?) was most interesting, though of course not at all peculiar to this particular form of lepromatous leprosy. (These were probably monocytes. M.S.)

J. A. KINNEAR BROWN, M.D., Entebbe, Uganda:

My opinion must be of limited value because we do not see this type of patient in East Africa. There appears to be little doubt from your protocol that you are dealing with a patient with lepromatous leprosy. I think the Lucio phenomenon comes into the differential diagnosis but, without seeing the patient, to say more would be rather difficult. I only wish I could be of more assistance.

E. MUIR, M.D., Middlesex, England:

I should very definitely confirm the diagnosis of diffuse lepromatous leprosy. The case corresponds with some I have seen in both India and Trinidad. It corresponds fairly closely with the description of the "Lucio phenomenon" described by Latapí and Zamora, and of which they say that some 15% to 20% of their cases in Mexico consist.

Why this type of case should be so much more common in Mexico than elsewhere it is difficult to fathom. One would naturally think of it as a racial peculiarity, corresponding with the racial leprosy distinctions found among the Chinese in Malaya. But your patient, a Negro, differs widely as regards race from the patients in Mexico. In the photos, the skin color seems light for a Negro. Could this subtype of leprosy be connected in any way with contrasting racial mixtures? (This seems not to be the case in the reported examples. V.J.D.)

Otherwise, have septic complications (gums, former injury with septic complica-

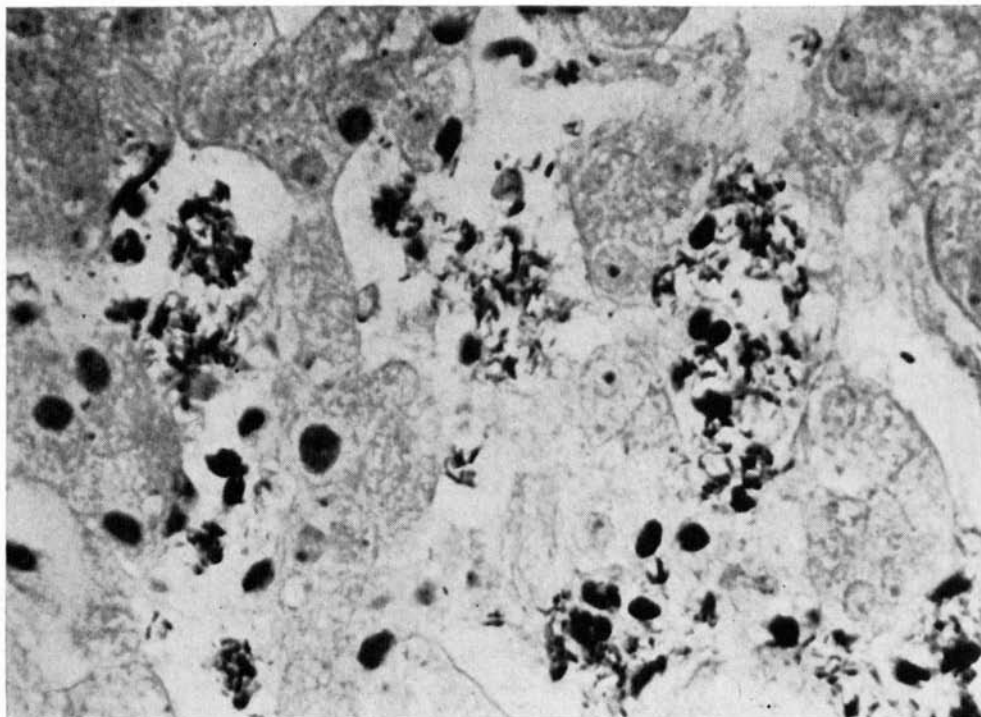


FIG. 10. Liver. Acid-fast bacilli seen in the Kupffer cells in the liver sinusoids. Fite acid-fast stain.

tions), or malnutrition, or syphilis, anything to do with it? (No, as far as can be determined. V.J.D.)

One interesting finding of Latapí and Zamora was that these cases made very rapid improvement under sulphone therapy.

MAXIMILIAN E. OBERMAYER, M.D., Los Angeles:

There is no doubt in my mind that the diagnosis of diffuse lepromatosis of Lucio is correct, clinically as well as histologically. This would then be the first case of the disease observed in a Negro. All other patients I have seen, in Mexico as well as the United States, were of the Mexican race.

V. PARDO-CASTELLO, M.D., Vedado, Habana:

I agree with your diagnosis. This case represents an immunological type of leprosy in which the patient has few if any allergic defenses against the infection; it is a septicemic condition as shown by the presence of innumerable Hansen's bacilli in the circulating blood, in the skin and the mucous membranes, as well as in the internal organs. The formation of flaccid bullae and of local necroses in the skin, very rich in bacillary content, is typical of these cases.

H. W. WADE, M. D., Palawan, Philippines:

The material which you sent me has been examined with great interest, especially as I am personally very ignorant of the Lucio condition.

It was completely unknown until Latapí *et al.* brought it to attention at the Havana leprosy congress in 1948, and then it was supposed to be found only in Mexico and Costa Rica. Since then a couple of cases have been reported in the United States (Los Angeles and Honolulu).

For me it was unknown before and after 1948, except that, during the Madrid congress in 1953, Lavalle, a confrère of Latapí's pointed out to Spanish leprologists some

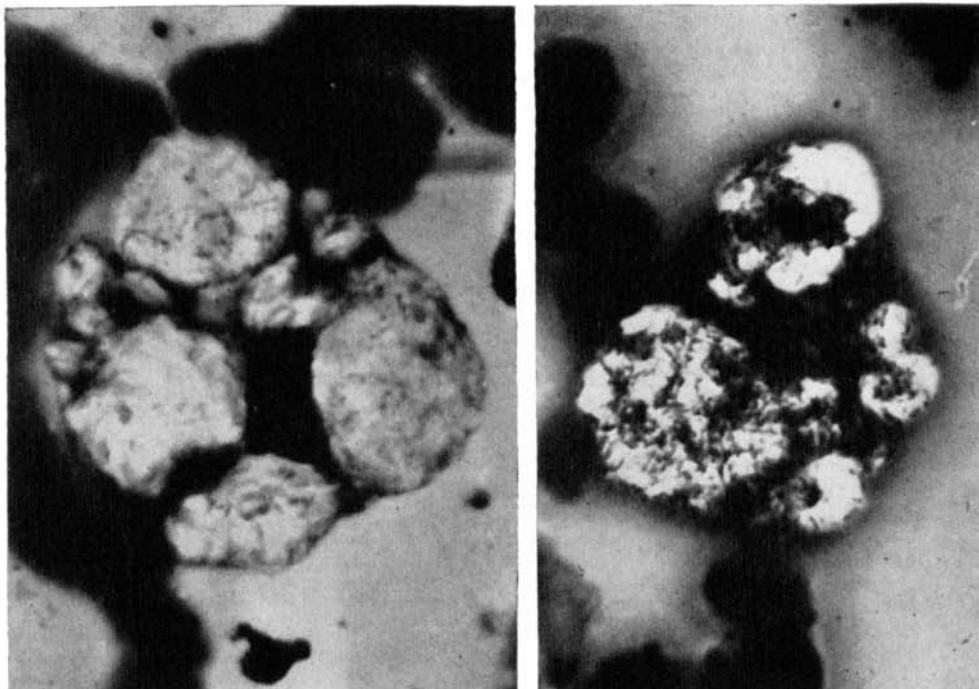


FIG. 11. Bone marrow aspirate. Typical "foam cell" seen in large numbers throughout the marrow smear. Wright's stain.

FIG. 12. Bone marrow. "Foam cell" containing myriads of acid-fast bacilli. Acid-fast stain.

cases which he asserted had had Lucio because of the nature of the scars. I was shown some of those cases and was—and am—dubious.

On the way back from that meeting, however, I visited Kalaupapa and saw there a patient with lesions which to me spelled Lucio. A biopsy specimen was promised and came along, but it was not from an ulceration. The histologic picture has no real resemblance to the one you have sent out. (For my own record I have made a few notes about them, and a copy is enclosed.)

Your protocol and pictures were referred to Dr. C. B. Lara, chief physician here for many years, thinking perhaps he may have seen such cases. (As pathologist, I do not see much of the clinical work.) He was at Havana and heard Latapí, but it is evident he does not know the condition from personal experience.

There are a few comments on your protocol notes that I take the liberty of making.

The chap was perhaps scared when examined to have a pulse rate of 128. Did that condition persist? Could he perhaps have been in a state of lepra reaction? (Yes. V.J.D.)

Loss of eyebrows is, of course, a characteristic of advanced lepromatous leprosy (loss of the eyelashes less common). But your man, from his pictures, seems far from a typical advanced lepromatous case. Is loss of eyebrows also typical of Lucio? (Yes. Latapí.)

About the thinning of the hair over the occiput, was there evidence of lesions of the scalp? (No. V.J.D.) Involvement of that region is rare except in Japan; but otherwise, why the loss of hair in that region? (Cranial alopecia has been reported in diffuse leprosy by Escalona and González Urueña. Latapí.)

In ordinary lepromatous leprosy there would be, along with the loss of eyebrows, more or less marked thickening of the earlobes. Nothing is said of that, only of the

finding of bacilli in smears. If not thickened, that would be noteworthy. (They were not thickened. V.J.D.)

Ulceration of the hand or elsewhere in ordinary lepromatous leprosy would never extend to the tendons except from neglected secondary infection, and a phagedenic one at that. Worth a bit of emphasis, no?

The pictures show zones of hyperpigmentation around scars.

Biopsies of the liver and kidney? Needle biopsies, presumably. (Yes. V.J.D.) Never done before to the kidney of a leprosy patient, I shall wager!

Altogether, perhaps the most thoroughly laboratory-investigated case of leprosy ever. The results of the examination of the blood for bacilli are particularly interesting; I wonder whether it was ever done before in a Lucio case. It is not difficult to find them in lepromatous cases in *lepra reaction* but not otherwise.

But one thing: *Not*, I submit (and this is a special field of mine), the *lepromin* test! Lepromin was injected, yes, but the record indicates observation, not for the late Mitsuda phenomenon, but only for the early Fernandez reaction. The late one would have been negative in this case, of course.

But here is the peculiar thing: according to Latapí injection of lepromin in Lucio cases elicits a very peculiar reaction, which he calls the Medina reaction. There is an early reaction, he says, which lasts for 24-48 hours, but it differs from the Fernandez reaction in that it is 'always frank and intense within the first 4 to 6 hours,' and looks and progresses like the Lucio phenomenon itself even (with sufficient dosage of lepromin) to necrosis ('expulsive, triangular ultimate necrosis,' our translator has it). Almost identical results can be obtained, they say, with injections of streptococci or staphylococci.

The larger section is from intact skin (of the arm) since the epidermis is continuous, although perhaps in danger of breaking down later. The highlights include a curious mixture of acute and chronic effects.

The papillary layer is peculiarly solid and structureless; the fibrous strands in the reticular zone are coarse but orderly; and below the dermis proper, separated by a strand of fat but connected, there is fibrous band reminiscent of chronic erythema nodosum leprosum. This zone shows fibrin infiltration, but much of it has the appearance of fibrinoid necrosis. The fat shows lepra-cell invasion in a fashion typical of lepromatous leprosy.

In the acute affection of small blood vessels (the "vascularitis" of the "erithema necrosante," Latapí) is the most conspicuous and interesting feature. It is well seen in the fat-zone infiltrate, which at one end is badly disturbed by superimposed acute infiltration while at the other end it is practically free of it; yet even there is found necrosis of the small vessels and consequent effects in the lepromatous infiltrate (diapedesis, etc.). Apparently, the vessels are damaged first, and the poly-infiltration comes later. In acute areas there and elsewhere are seen tortuous large mononuclears writhing between the other cells, in a most unusual fashion.

A few blood vessels are seen with circumferential adventitia infiltrated by mononuclear cells, without polyps, after the fashion of infiltration of the perineurium of small nerves of the dermis in tuberculoid leprosy. There is an evident tendency for red cells inside these vessels to agglutinate against the walls.

A striking and utterly peculiar feature of the chronic phase is a perifibrosis of many of the small vessels, not a part of the normal picture of leprosy and new to me. When I first looked at one end of the section I noted a group of three structures with circumferential fibrous layers very like the flaky, "pie-crust" proliferation of the perineurium of small dermal nerves that is highly characteristic (but not absolutely pathognomonic) of the lepromatous type of lesion, but there was a double-take when it was seen that one whorl was centered by an open vessel lumen. In it are central proliferative changes that may account for the evident obliteration of the channel (the solid condition) of the other two structures. (A Mallory aniline-blue section would be useful here, since it demonstrates well small bundles of endoneurial fibers in places where nerves would

otherwise be overlooked or very difficult to identify, and they should be present centrally in these structures if they actually are nerves.) However, from other examples in that section, and several in the smaller section, I believe they are all derived from small vessels, especially since two or three definite nerves show no perineurial proliferation. (They are, however, infiltrated and would doubtless show many bacilli.)

Unexpectedly in view of the pictures, there are a few small active foci even in the eyebrow section. (How much of the surface of this chap would show 'inapparent' involvement?) (All. V.J.D.)

The picture as a whole is unlike any seen before.

ROLLA R. WOLCOTT, M.D., Carville, La.:

I have reviewed your protocol and have talked with the patient here. I offer the following comments:

The salient points in the history, physical examination, and laboratory findings are the following:

1. This 52-year-old Negro male patient has spent his entire life in Louisiana, approximately 100 miles north of the area generally considered to be the more endemic part of the state.

2. First symptom suggestive of leprosy was chronic nasal obstruction first noted in 1948.

3. Subsequent complaints suggestive of leprosy were loss of eyebrows and eyelashes, anesthesia of extremities, ulcers of the extremities, and progressive weight loss.

4. Physical examination shows madarosis, atrophic rhinitis with crusting, widespread diffuse skin infiltration of slight to moderate degree, near total anesthesia of the extremities from elbows and knees distalward, and numerous punched-out ulcers of the extremities.

5. Laboratory findings include the presence of *M. leprae* in skin and nasal scrapings, reversal of albumin: globulin ratio, 4+ cephalin flocculation, reactive Kahn and VDRL tests.

6. Lepromin test is negative.

The above picture presents to me a classic example of la Lepra "Manchada" de Lucio, originally described by Lucio and Alvarado in 1852 and more recently described by Latapí and Chevez, who speak of it as pure, diffuse, lepromatous leprosy with Lucio phenomenon.

FERNANDO LATAPÍ, M.D., México, D.F.:

I have observed the histopathologic slides with the photographs and I agree that we are dealing with diffuse lepromatous leprosy (Lucio leprosy).

This type of leprosy abounds in Mexico and it is well known to us. I am sure that in the south of the United States there are probably many more patients of this type.

FELIX CONTRERAS, M.D., Madrid, Spain:

I take pleasure in giving my opinion together with that of Prof. Gay Prieto, who has seen the kodachromes and biopsies of your interesting case, and agrees with us in full, as I shall explain.

The excellent photographs in color give us the impression that this is an example of nodular lepromatous leprosy. with some of the lesions ulcerated, a variety which we see with relative frequency among our patients.

As the 'Lepra de Lucio' is a diffuse infiltrative form in which Lucio underscored the absolute absence of nodules we do not believe that your interesting case of lepromatous leprosy can be classified in the mentioned variety. (There were no nodules in this patient. V.J.D.) Moreover, in the detailed history which you sent me, we see that your patient had a negative lepromin at 24, 48, and 72 hours. This also contradicts the concept that we are dealing with a form of Lucio because Medina described in these cases a special reaction to lepromin, positive; frankly and intensely, from the first hours, and should not be confused with the Fernandez reaction because staphylococci and streptococci antigens may produce the former.

I am sending you a copy of what we see in your histologic preparation, which in essence coincides with your opinion, since we established the existence of necrosis, which might be the reason for orienting the diagnosis to the Lucio form.

With all these facts, and without being able to give an affirmation, since only you, with the patient before you, can really decide, yet we believe that you may be dealing with a case of nodular lepromatous leprosy, in a reaction in which phenomena of vasculitis and necrosis predominate. We see patients in these same conditions which we do not dare to subclassify in the form of Lucio.

Following is the enclosure from Dr. Contreras:

Epidermis.—Atrophy. Moderate hyperkeratosis. Extremely abundant melanin in the basal layer. Subepithelial zone intact.

Dermis.—Abundant infiltrates with localization predominantly perivascular, periglandular, and around the hair follicles. These infiltrates are typical lepromatous granulomata composed of lymphocytes, Virchow cells together with fibroblasts, and histiocytes, but there are no foreign body giant cells. Some of the nuclei of these cells present in the preferentially perivascular infiltrates signs of necrosis, for example pyknotic nuclei as well as karyolysis and more rarely karyorrhexis. One also sees perivascularly some infiltrates made up of abundant polynuclear lymphocytes and tissue cells. In a few of these infiltrates one observes signs of necrosis.

The majority of the vessels are affected, some presenting sclerosis of the walls, and others showing hyaline thrombi. There are evident signs of the existence of a previous new formation of vessels, but in the slide which we are describing many of these vessels are in process of necrosis.

In the hematoxylin-eosin stain the collagen fibers may be seen to be in large part swollen and compressed, staining homogeneously in masses which are essentially anuclear but which do contain nuclear remnants. This portion speaks in favor of a fibrinoid degeneration.

The hair follicles and sebaceous glands are in good condition, as are the sweat glands, except in those regions where the infiltrate is extremely dense, where they appear slightly atrophic.

Hypodermis.—The same structures are observed as those seen in the dermis, especially in the nonspecific perivascular infiltrates. Infiltrating the interstices of the fatty tissue are elements of lepromatous granuloma assuming the typical disposition in the mesh form. The picture of nonspecific perivascular infiltrate abounding in polynuclear cells recalls at times the picture seen in erythema nodosa.

SUMMARY AND CONCLUSIONS

A case of Lucio phenomenon leprosy is presented. The patient is an American Negro who has never been out of the state of Louisiana. The salient features of this form of the disease include loss of eyebrows and eyelashes, anesthesia of extremities, "manchas" or erythematous spots which go on to vesiculation, superficial necrosis and scarring, atrophic rhinitis, widespread diffuse skin infiltration, absence of lepromata, presence of *M. leprae* in skin, nasal scrapings, and blood, reversal of albumin-globulin ratio, and false-positive serologic tests for syphilis. The opinion of world famous leprologists was sought by correspondence, and it was the consensus that this example represents a valid instance of diffuse lepromatosis.

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PERSONAL COMMUNICATIONS

- ARNOLD, H. L., JR., M.D.: Straub Clinic, Kapiolani St. at Thomas Square, Honolulu 14, Hawaii.
- BROWN, J. A. K., M.D.: Specialist Leprologist Medical Department, Headquarters Office, P. O. Box 8, Entebbe, Uganda.
- CONTRERAS DUEÑAS, F., M.D.: Médico Director de Fontilles, Moreto 15, Madrid, Spain.
- MUIR, E., M.D.: Oakhurst, 29A Crawford Ave., Wembley, Middlesex, U.K.
- OBERMAYER, M. E., M.D.: Clinical Professor and Chairman, Section of Dermatology, University of Southern California School of Medicine, Los Angeles 33, Calif.
- PARDO-CASTELLO, V., M.D.: Catedra de Piel y Sífilis, Universidad de la Habana, Habana, Cuba.
- WADE, H. W., M.D.: Leonard Wood Memorial, Culion Sanitarium, Palawan, Philippines.
- WOLCOTT, R. R., M.D.: Medical Director, USPHS, Clinical Director, Public Health Service Hospital, Carville, La.