LEPROSY AND SYPHILIS

By

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About three months ago, a woman with a skin disease (Figs. 1, 2, 3) came to our clinic. This disease had caused a boarding house to refuse to hire her as a cook.

She was a Brazilian, single, a Negress from the State of Minas Geraes. She claimed to be twenty years old, although she looked older.

Previous health history, both of herself and of her family, was not important. Her mother and four sisters were apparently healthy. She asserted that there were no cases of leprosy in her family; indeed, she had never lived with any person having leprosy. She denied ever having had any venereal disease.

In discussing her present disease, she mentioned that some pale blemishes had begun to appear on her face, about eight months prior to the date of the consultation. These blemishes gradually darkened. About a month after the blemishes were first noticed on her face, another large blemish, of the same appearance, began to show on her right buttock; after this, some others, small and large, began to appear all over her body. An objective examination revealed that she was in good general condition. On her forehead, right cheek, chin, and upper lip, she had large, circinate patches of various sizes, somewhat infiltrated, marginated by miliary tubercles. The center of some of these patches was lighter in color than the patient's uninvolved skin. In most of the patches, however, the center was clearly darker, and copper-colored. The patient's ear lobes and the tip of her nose were also infiltrated, and copper-colored as well. There were many of the circinate patches to be seen on her torso, and on her upper and lower extremities. Some of the patches were annular, not infiltrated, with an atrophic, light-colored center, marginated by small, refringent tubercles, in the shape of a darker ring. Other patches were light in color in their entirety. The hair of the eyebrows was thin. No thickening of nerves was apparent to the touch; there was only slight swelling of the inguinal lymph nodes. There was no muscular atrophy. In the facial lesions, sensitivity was normal; in the other lesions, there was decreased sensitivity to heat and pain. In others, for instance, one on the patient's right arm, there was practically no sensitivity to pain, and only a retarded reaction to heat. In all lesions, however, the sensitivity to touch was normal. In order

PLATE I

FIG. 1. Face of the patient. On the forehead, right cheek, nose, and chin are circinate patches of various sizes, slightly infiltrated, marginated by miliary tubercles. The center of some of these patches is hypopigmented; in most, however, it is hyperchromic, copper-colored. Normal sensitivity.

FIG. 2. Lateral view of the right arm, showing hypochromic patch, plane, circinate, with an atrophic center, marginated by small, refringent tubercles, in the shape of a darker ring. Absence of sensitivity to pain; retarded reaction to heat and normal sensitivity to the touch.

FIG. 3. Full view of patient showing widespread occurrence of the lesions.

FIG. 4. Photomicrograph (X approx. 180) of sarcoid granuloma with two vessels. The whitish elements, more numerous, are epithelioid cells; the brownish, smaller ones are lymphocytes. The endothelium of the inner wall is thickened; the round muscular cells of the media are perpendicular.

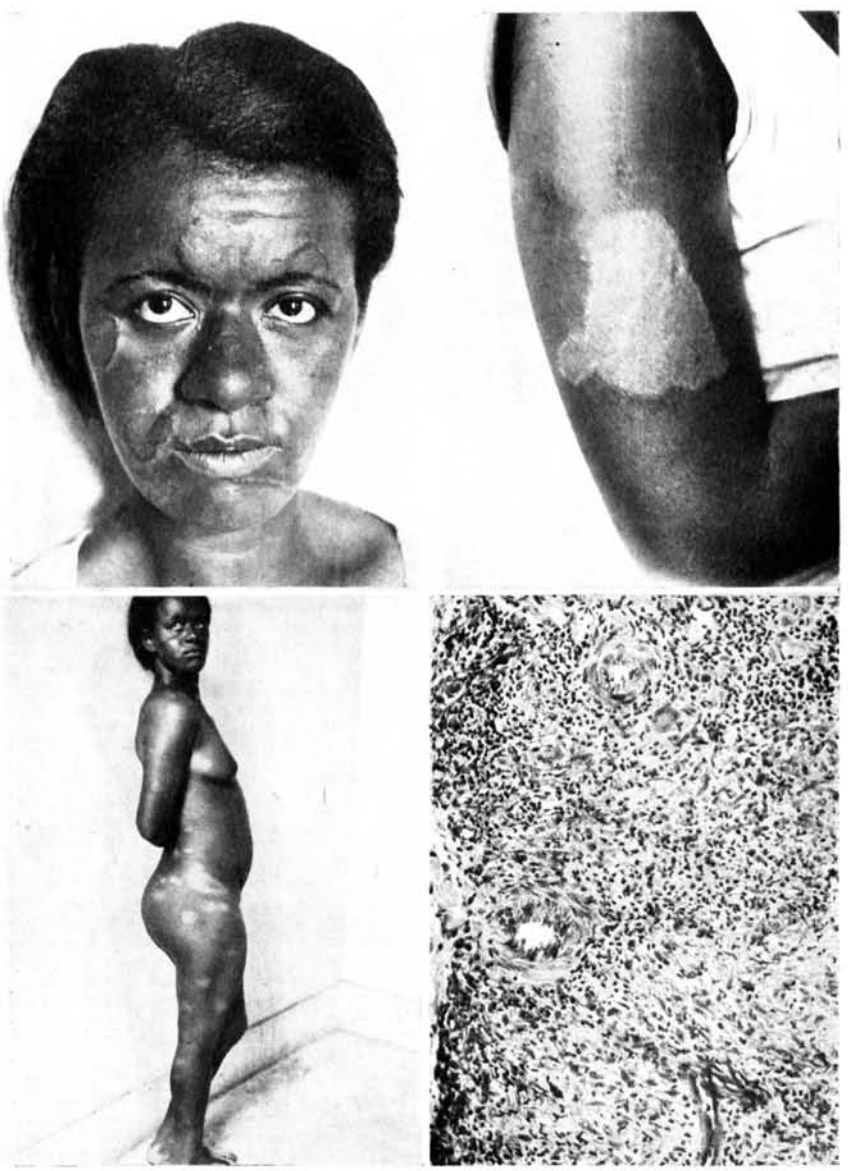

PLATE I

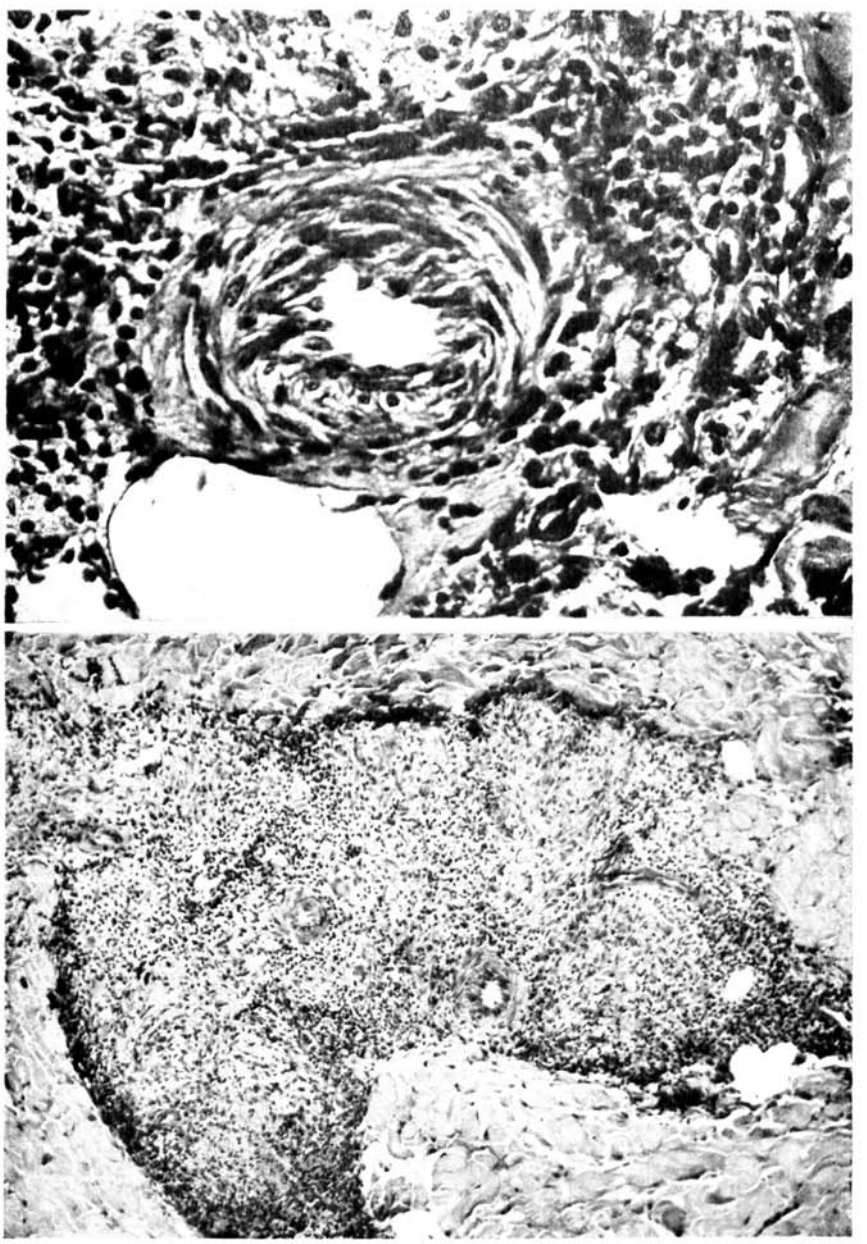

Plate II

PLATE II

FIG. 5. Photomicrograph (X 150) The lighter field is formed by epithelioid cells, marginated by smaller and darker elements, and lymphocytes, which are also seen in the center. Three vessels, with parietal lesions, are included in the infiltration.

FIG. 6. Photomicrograph (X 450) Thickening of the endothelial wall of a vessel with muscular wall, causing narrowing of the vessel. In the upper part, epithelioid infiltration; at the sides, lymphocytic infiltration; below, a fat cell.

to complete our observations, we decided to intern the patient in a ward. The ophthalmological examination disclosed the presence of mucous conjunctivitis and palpebral exfoliation. *Mycobacterium leprae* was not found in the nasal mucosa, nor in an inguinal lymph node, nor in the smear from one of the lesions.

Sinapisms were applied on normal and diseased skin. Erythema with itching was noted in all lesions. On the normal skin, however, the itching was much more intense. The histamine test, in a 1/500 solution, showed lack of the second erythema on all lesions on which it was made, on the face as well as on the rest of the body. On the healthy skin, Lewis's three answers were observed. Two biopsies were made from lesions, one from the face and another from the left forearm.

The result of the histopathological examination follows:

"Some foci of espongiosis and exocytosis are found in the epidermis. There are large cellular infiltrations in the corium, separated by normal connective tissue. Some of those infiltrations are localized in the papillary layer, and come in contact with the epidermis where the above-mentioned alterations of the epidermis are observed. Other infiltrations, found in the medium or deep corium, are of round or cylindrical shape, and arranged either vertically or horizontally, toward the hypodermis, but not reaching it. Hairs and glands are generally involved in the infiltrations, which have usually a uniform composition of epithelioid cells in great numbers, and lymphocytes. In a few places, Langhans' giant cells are seen. In the middle of the infiltrations are seen vessels with muscular walls with thickened endothelial walls, causing narrowing of the lumens.

"In another section from the same patient, in addition to the infiltrations of identical structure as those of the first biopsy, some others are seen, constituted almost exclusively of lymphocytes. In these infiltrations, as well, the blood vessels show the same changes." (Dr. Hildebrando Portugal).

The observed structures (Figs. 4, 5, 6), as we can see, are characteristic of the sarcoid granuloma; they do not allow us, therefore, to make any assumptions—without additional information—as to the etiological agent concerned.

Wassermann's and Muller's blood tests were positive.

With such clinical, serological, and histopathological data, we believed this to be a combination of syphilis and leprosy. We decided to begin the treatment of syphilis, excluding from the first the arsenic preparations, the use of which is not advisable for Hansen's disease. It was necessary to find a medication which would succeed against one infection without increasing the other. We tried a bismuth compound, soluble in oil, which was used for three weeks, with a total of 33 cc.

of metallic bismuth. Considering that this preparation had been tried in our wards with good results, we considered it strange that we did not notice even the slightest improvement in the patient's condition. We then began the anti-leprous therapy, using iodized (0.5%) ethyl ester of chaulmoogra, with one injection of 5 cc., one of 7 cc., and seven of 10 cc., all intramuscularly. This course of treatment was carried on over a three-week period. There was no improvement under this treatment; the appearance of the lesions was not changed.

We then decided to use a more intense anti-syphilis treatment, through the use of neoarsephenamine, intravenously. Following the initial doses the patient was carefully watched, in order to observe whether the lesions would get worse. All injections were well tolerated. No lesion increased—on the contrary, the objective examination showed considerable improvement. Up to now the patient has been injected with 4.95 grams of neoarsephenamine, and all facial lesions show less thickening although the copperish-brown coloring still persists. In the lesions of the rest of her body the thickened edges are beginning to disappear; the center of the lesions, which was definitely pale before, is beginning to appear like normal skin. The decreased sensitivity, however, was not modified by the treatment, that is, it persists in the facial lesions and continues lessened on the others. In the face of these facts, what shall be our diagnosis? It seems to us that there are three hypotheses to be considered: syphilis, leprosy, or an association of syphilis and leprosy. If we choose the first hypothesis, we should classify the case as one with secondary-tertiary manifestations, considering the appearance of some of the lesions (such as those on the face), the positive blood test, the endarteritis revealed by biopsy, and, finally, the "ex-juvantibus" evidence, that is, the real improvement caused by arsenotherapy. The vascular lesions are in favor of a diagnosis of syphilis. There would be, however, some difficult questions, if we adopted this diagnosis. Could we envisage a peripherial neuritis caused by syphilis and responsible for hypoesthesia and for the absence of the second erythema in the histamine test? Is not the sarcoid infiltration very rare in syphilis?

The second hypothesis would refer to a tuberculoid type of leprosy. If anyone experienced in examining leprosy patients were to study our patient—with the possible exception of some of the facial lesions—he would not hesitate for a moment in reaching this diagnosis. The morphology of most of the patches is typical; in addition, some of the patches are hypoesthetic. The second erythema of the histamine test is lacking, however; moreover, leprosy does not cause vascular lesions. Why did the arsenic not increase the infection? Is it possible that the tuberculoid type of leprosy is not made worse by this medication?

We believe that there are strong arguments in favor of the hypothesis of a syphilis-leprosy hybrid. There are some lesions which are

like syphilis such as on the face, with copper-like discoloration, and normal sensitivity; on the rest of the skin, there are patches which are typical of tuberculoid leprosy, hypoesthetic, hypopigmented, and with central atrophy. The histopathology corroborates this impression, showing the coexistence of the sarcoid structure, and frequent and uniform vascular lesions on both biopsies. It is at this point, we believe, that the greatest interest of this observation lies. In pathology, especially dermatological pathology, histology must have the last word.

The clinical examination and the blood tests explain satisfactorily the simultaneous presence of both lesions. The patient had symptoms of leprosy (alterations of sensitivity) responsible for the sarcoid granuloma—as well as positive Wassermann and Muller tests, indicating syphilis, which caused the vascular lesions.

We intend to proceed with the antisyphilitic therapy, with use of a bismuth compound, as soon as the first series of neoarsephenamine is finished. Finally, if the lesions do not disappear completely, and we believe that they will not, we will try chaulmoogra oil again, in larger doses. We hope to confirm the old aphorism, "Naturam morborum curationes ostendunt."