EXACERBATION OF LEPROSY DURING PRESENT DAY TREATMENT [1]

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Experience with a variety of sulfone and other compounds at Carville since 1941 has shown that the majority of treated patients respond with subjective and objective improvement. The degree of bacteriologic improvement does not keep pace with clinical improvement. The result after years of therapy is often a rather stable leprous process of low-grade activity. In a very small number of cases improvement goes on to a state of apparent arrest in which there is not any evidence of clinical activity and *M. leprae* cannot be demonstrated by ordinary means. Until 1948 severe exacerbations of leprosy were not observed among patients receiving treatment at this hospital.

It is the purpose of the present paper to present briefly, by case histories and clinical photographs, a few examples of the more serious exacerbations of leprosy which have occurred during the past five years in hospitalized patients during sulfone and other current treatment regimens.

CASE 1.—This white male patient was born in 1904. The onset of his illness occurred in 1927, and he was admitted to Carville in 1935 with moderately advanced lepromatous leprosy. At time of admission he weighed 137 lbs.

The lepromatous lesions remained relatively stationary during several years of chaulmoogra oil therapy. The photograph shown in Fig. 1 was taken in 1940 after five years of chaulmoogra oil. The patient became subjectively and objectively worse during several months of diphtheria toxoid injections.

Intravenous promin therapy was started in the summer of 1942, the patient then weighing 145 lbs. Between then and 1952 a total of 4,284 gm. was given. During the latter half of 1942 the patient experienced his first attack of erythema nodosum leprosum, with fever and ulnar neuritis. After 1944 erythema nodosum did not occur. Atrophic scarring of skin lesions over the trunk is to be seen in a photograph taken in 1946.

[1] Read at the VI International Congress of Leprology, Madrid, October 1953. Modified in that the original paper referred to numerous slides shown on the screen, whereas only a limited number of the pictures are reproduced here.

Clinical improvement continued, and in November 1949 eight consecutive monthly skin scrapings from various parts of the body had failed to reveal *M. leprae.* Examination in November 1949, however, revealed large numbers of *M. leprae* in new lesions on the left buttock and the right elbow. Fig. 2 shows the appearance of his face at that time.

Since the latter part of 1949 this patient's lepromatous leprosy has shown a marked exacerbation during periods of sulfone and isoniazid therapy. Fig. 3 shows the extent of the lesions of the face in June 1953. Ulceration had occurred in many of the lepromata on the elbows and buttocks. All lesions contained innumerable *M. leprae.*

In spite of the marked extension of his disease, this man's general health apparently has not suffered. His body weight in June 1953 was 188 lbs.

CASE 2.—This white male patient was born in 1909. The onset of his illness occurred in 1939, and he was admitted to Carville in 1940 with lepromatous leprosy. At the time of admission he weighed 141 lbs.

Chaulmoogra oil was given from 1940 to 1944, and eight injections of diphtheria toxoid were given during 1941 and 1942. Erythema nodosum leprosum with fever appeared for the first time September 18, 1942, during the administration of promin. Sulfone drugs were continued from 1942 to 1952. After 1946 he did not have any further acute attacks of erythema nodosum. This man's clinical improvement was satisfactory during the period 1944 to 1950.

By July 1950 eleven monthly examinations had failed to show *M. leprae* in skin scrapings. At the examination made then a new conglomerate group of lepromatous nodules of keloid consistency were seen in the left epigastric region, and scrapings from this area showed large numbers of *M. leprae.*

From 1950 to 1953, during which time he was under thiosemicarbazone, isoniazid, dihydrostreptomycin and PAS regimens, this patient showed a wide extension of new lepromatous lesions over the entire body. His general health, however, did not suffer during this period, although he experienced great emotional strain due to worry about the extension of his disease. His weight in July 1953 was 204 lbs.

CASE 3.—This white male patient was born in 1925. The onset of leprosy occurred in 1939, and patient was admitted to Carville in 1941 with rather early lepromatous leprosy. There was some thickening of the left ulnar nerve, but no contractures.

Treatment included promacetin from 1942 to 1945, promin from 1946 to 1948, and small amounts of sulfone drugs, streptomycin, PAS, tibione, aureomycin and other medications from 1948 to 1951.

This patient improved in general health and his specific lesions decreased during the period 1942 to 1945, while he was experiencing repeated attacks of erythema nodosum. Fig. 4 shows his condition in 1945. From 1945 to 1948 he had less erythema nodosum. In the latter part of February 1948 he began to have symptoms of ulnar neuritis of both arms. For a period of approximately 15 months he suffered exquisite pains of the extremities and was confined to bed. By the latter part of 1949 there were severe contractures of the hands. During 1950 and 1951 the patient suffered additional nerve damage but was ambulatory much of the time. In November 1951 he left the hospital with active lepromatous leprosy. He took only small amounts of sulfone drugs at irregular intervals during

1951 and 1952 and was not seen until he returned to the hospital in April 1953 because of further acute extension of nerve and skin involvement. His clinical picture during April and May 1953 is shown in Figs. 5 and 6. Since 1948 his body weight has ranged from 135 lbs. when ambulatory and free from pain to 100 lbs. or less during long periods of confinement to bed.

In recent years a number of instances of rapid extension of the leprous process have occurred in Carville patients while they were receiving continuous sulfone or other treatment. The disease had usually reached a stage of apparent quiescence before the occurrence of new symptoms and new lesions.

Careful observation and study of patients with exacerbations have failed to define any common factor which might have precipitated the aggravation of their disease. In most cases the patients had enjoyed a state of good general health and their body weight had been maintained. Intercurrent disease had not been present. Laboratory studies had not shown any unusual abnormalities suggestive of relapse. A few individuals had experienced some increase of emotional strain prior to onset of new lesions.

In those patients with lepromatous leprosy, erythema nodosum had usually disappeared from the clinical picture several years before the recrudescence of the disease.

SUMMARY AND CONCLUSIONS

1. Since 1948 exacerbations of leprosy have been observed in hospitalized patients receiving sulfone and other present-day treatments.
2. Case histories and clinical photographs illustrate typical cases of aggravation of lepromatous leprosy.
3. The cause of the exacerbation is not apparent.
4. Erythema nodosum is usually absent before exacerbation occurs.
5. The possibility of clinical and bacteriologic exacerbation must be considered in evaluating the prognosis of patients undergoing present day treatment.

RESÚMEN

Desde 1941 hasta el 1948 se han tratado muchos pacientes con las drogas sulfonas y otras, sin observarse recrudecencias en aquellos casos mejorados por las drogas. Desde el 1948 se han observado varios casos los cuales desarrollaron síntomas de reactivación durante el curso de tratamiento con sulfonas. En estos casos no se observó eritema nodosum antes de la exacerbación. No se ha podido determinar la causa de éstas reacciones. Se debe considerar la posibilidad de una exacerbación clínica y bacteriológica, en la evaluación del prognóstico de pacientes bajo tratamiento con las drogas actuales.

DESCRIPTION OF PLATE

PLATE (10)

FIG. 1. Case 1, showing the condition of the face in 1940, after five years of chaulmoogra therapy.

FIG. 2. Case 1, showing the improvement in 1949 after several months of bacterial negativity, at the time that new and positive lesions were found on other parts of the body.

FIG. 3. Case 1, showing the lesions of the face in 1953, more than two years after relapse.

FIG. 4. Case 3, showing the face in 1945, during the period of improvement.

FIGS. 5 and 6. Case 3, showing the condition in 1953, about five years after symptoms of acute neuritis first appeared.

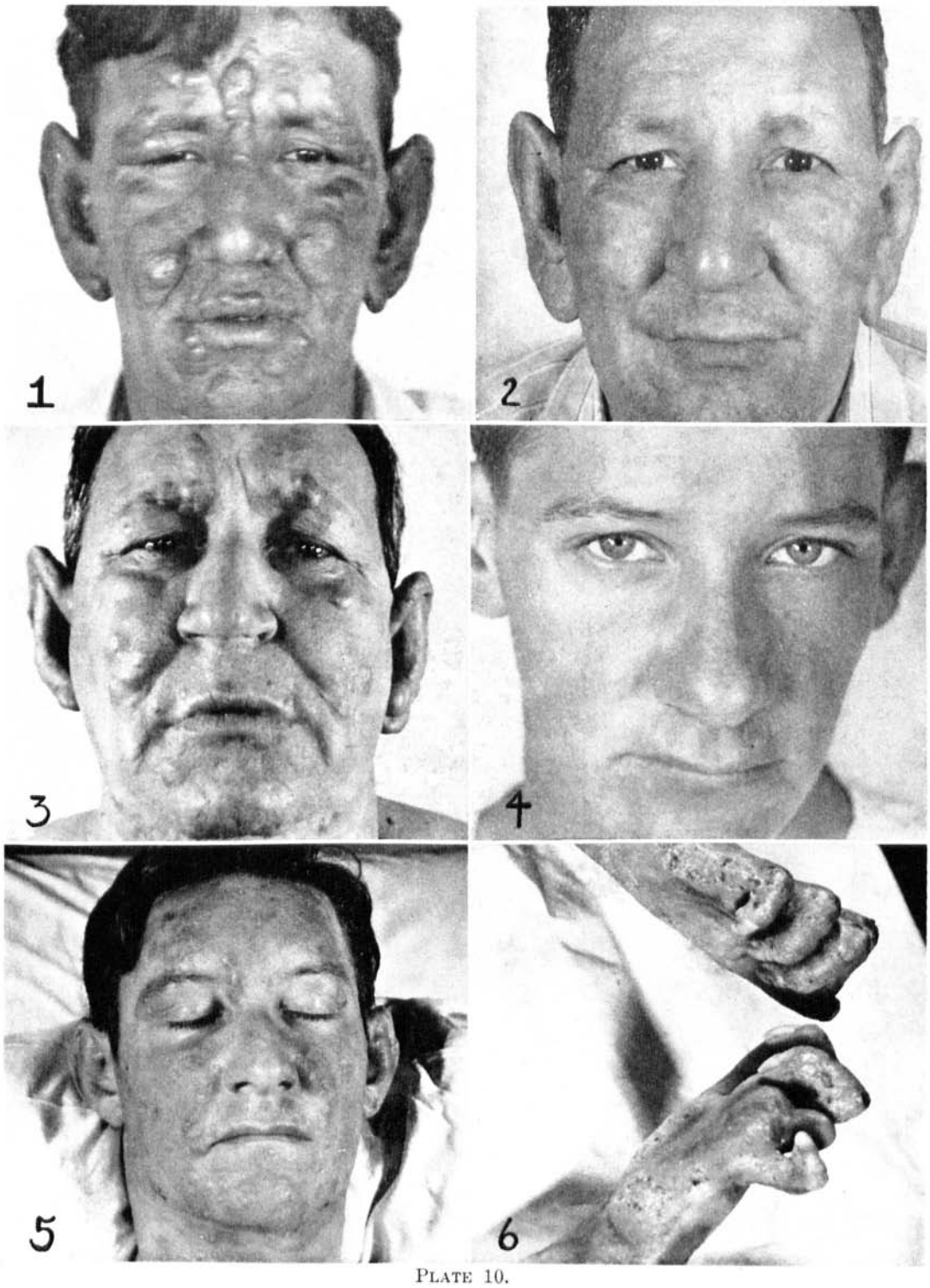

PLATE 10.