NOTES ON A CASE OF LYMPHADENOMA COMPLICATING LEPROSY

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This case of lepromatous leprosy is of especial interest because no leprosy bacilli could be found in the skin during the last two years of the patient's life. Death resulted from lymphadenoma (Hodgkin's disease).

It may serve also as a good review of the methods and results of treatment at the Pretoria Leper Institution.

Methods of treatment. In neural cases that are not in the reactionary phase chaulmoogra oil is given by subcutaneous and intradermal injections up to an amount approximately 1 cc. per 10 pounds of body weight every week. The subcutaneous injections of iodized ethyl esters of chaulmoogra are given bi-weekly in doses of from 2 to 5 cc. and are regarded as systemic treatment. The intradermal injections are also given bi-weekly in doses up to 8 cc. This treatment is regarded as local treatment of the maculae and causes a relatively speedy resolution of the maculae. Experiment has shown that if only one half of the body is injected, the untreated areas remain unaffected. Local treatment, however, is more than cosmetic as Lie (1) and Ermakova (2) have shown that bacilli are present in the nerve fibrils leading into maculae. Local intradermal treatment is a direct assault on these bacilli.

Results of treatment. The results of treatment are that from 20 to 30 per cent of the neural cases are discharged each year. Before considering a case for discharge the Annual Leprosy Board must be satisfied that no bacilli have been found for a minimum period of 12 months. At least two smears are examined each month, either nasal smears or skin smears, which are taken according to the incised skin scraping of Wade. (Under such routine examination 80 per cent of the neural patients do not show bacilli throughout their institutional history.)

The Board must also find no external evidence of clinical activity or report of any recent appearance of new maculae.

The treatment of lepromatous cases is a very different proposition and after eighteen years trial of various dosages and methods of chaulmoogra administration the conclusion has been reached that lepromatous leprosy is almost never cured. Temporary improvement and regression of lesions do occur but invariably bacilli can be found in the skin. It is, of course, a common experience to find regression of leprotic lesions in a patient who is

dying of a wasting disease such as pulmonary tuberculosis, but always in such lepromatous cases bacilli can be found although repeated search may be necessary.

The case to be discussed (number 518) was in the Pretoria Leper Institution for four years and was paroled under home segregation rules while still discharging bacilli in his nasal secretions and harboring them in his skin. He was seen only at varying intervals after this so his progress records are unfortunately meagre in detail.

Case history: European male aged 47 years; admitted November 1937; stated that 5 years previously he had noticed his foot going to sleep. Three years later the first mark appeared on his left forearm and then on his right buttock. His mother had died of leprosy in January 1937.

Physical examinantion revealed the following: general condition—very good; skin—erythematous lepromatous plaques on face, limbs, and body, nodular infiltration of face, and commencing loss of eyebrows; anesthesia of hands on ulnar side and of left foot up to ankle; ulnar and peroneal nerves not tender, painful, nor enlarged; slight contraction of little finger of left hand; nose healthy; nasal and skin smears positive for *M. leprae;* Wassermann strongly positive.

Energetic anti-syphilitic treatment was given, that is 18.4 Gm. of neoarsphenamine (N.A.B.) and 40 cc. bismuth in 15 months. The Wassermann reactions were: 1937, 1938, and 1939, strongly positive; 1940, doubtful; 1941 and 1942, negative.

In August 1939, when he developed a severe neuritis, he received 45 cc. intravenous mercurochrome.

His leprotic condition remained stationary despite bi-weekly injections of chaulmoogra oil. In April 1939 a reactionary phase occurred in which new plaques and nodules appeared. This was treated with two courses of fouadin. The plaques faded but new nodules broke out both in June and July. These gradually subsided and one year later the report states: "—no obvious infiltration of face; dusky blush on left flank at site of old nodule; some erythema on right flank; *new nodules(?) inside left arm.*" These nodules in the left arm which were questioned were most likely the epitrochlear glands becoming palpable as a result of a lymphadenomatous affection.

The patient was discharged in April 1940 and readmitted in December 1941. At this stage his liver and spleen were grossly enlarged and multiple superficial glands were palpable. The pathologist's report on a specimen of tissue was: "Sections of this lymph node show the changes of a very cellular type of lymphadenoma with numerous mirror image giant cells but little evidence of fibrosis."

Owing to the great abdominal discomfort he was given deep x-ray therapy to the spleen at weekly intervals during January 1942. The radiologist reported: "Hodgkin's disease, complicating leprosy, splenomegalia (ten fingers below the costal arches); the spleen is harder than is usual in Hodgkin's disease. The following treatments were given:

Date	kv.	Filter	Distance	Dose	Size of field	Location
Jan. 5, '42	200	Lcu.	50 cm.	200 r.	15 cm. square	Ant. spleen
Jan. 6, '42	"	"	"	"	" "	Post. "
Jan. 12, '42	"	"	"	"	" "	Ant. "
Jan. 13, '42	"	"	"	"	" "	Post. "
Jan. 16, '42	"	"	"	"	" "	Ant. "

Any improvement from the x-ray treatment would occur within a week or two. If at any future date the patient's discomfort increases due to further enlargement of the spleen, more x-ray treatment would be indicated."

The spleen which had been 3 inches below the level of the umbilicus in January had receded 3 inches by March when he was again discharged at his own request. The clinical notes regarding his leprotic condition then said: "—no clinical activity; all skin smears negative."

The patient was not seen again and is reported to have died on May 11, 1942, of lymphadenoma (Hodgkin's disease).

Smear history:

November 1937	— nasal smear positive for *M. leprae*
" "	— skin forehead " " " "
July 1938	— skin forehead " " " "
January 1939	— skin forearm " " " "
April 1940	— skin smear " " " "
" "	— nasal smear negative for *M. leprae*
September 1940	— nasal smear " " " "
" "	— skin wrist " " " "
March 1941	— skin forehead " " " "
" "	— nasal smear " " " "
March 1942	— nasal smear " " " "
" "	— skin forehead " " " "
" "	— skin forearm " " " "

As has been stated, this conversion from persistently positive to persistently negative smears is unique in the experience at this leprosarium. This cannot be attributed to the treatment which the patient received, as his treatment was routine and these results have not been duplicated in other lepromatous cases. The diagnosis of lepromatous leprosy was confirmed for four years in succession by our Annual Leprosy Board. It may therefore be inferred that the lymphadenoma which developed either attenuated the leprosy bacilli or actually eliminated them.

References

(1) Lie, H. P. Demonstration of the leprosy bacillus in the leprids. Internat. J. Leprosy. 3 (1935), 473-476.

(2) Ermakova, N. The histopathology of simple leprids. Internat. J. Leprosy. 7 (1939) 495-508.