

INFECTIONS IN LEPROSY INSTITUTIONS

A. R. DAVISON, MRCS, Eng.; LPCP, Lond.
*Medical Superintendent, Westfort Institution
Pretoria, South Africa*

I have noted in recent years what I think to be dangerous concepts creeping in among leprologists. The first is that leprosy is only, or mostly, acquired in infancy; and, following on this concept, that it is unnecessary for adults to take even rudimentary precautions against infection. I will attempt to throw light on these concepts by first reviewing the literature of infections of members of the staffs at overseas institutions, and then by recording the infections that have occurred in South Africa. The possibility that some of the cases detailed were infected in childhood cannot be completely excluded, but the probabilities are that they were not. It is most unlikely that anyone would seek work in a leprosy colony if he knew of a case in his family.

The first recorded case that I can find of infection of a doctor was reported by Robinson in the *Lancet* in 1867. In the literature up to 1930 as surveyed by Klingmüller (1), a total of 16 doctor infections were recorded. This includes one who was infected in South Africa. I know of another case infected in this country.

Rogers and Muir (2) report 139 cases of infection among male and female nurses and other attendants of leprosy patients. I have found references in Klingmüller to 34 more cases. There are also references to 11 priests or ministers and 5 nuns who developed the disease. It seems beyond doubt that many cases of infection have occurred which have not been recorded. The tendency would be to hush such things up, rather than to bruit them abroad.

The first doctor to acquire leprosy in South Africa (G. T.) was born in Great Britain in 1848 and became medical superintendent of this institution in 1901, when he was 53 years of age. His duties included clinical work and postmortem examinations. He retired from here in 1907, going to Britain, and reported to the South African government in 1910 that he had developed leprosy. Definite information as to the clinical nature of the disease has not been obtained, but it is understood that it started as of the "anesthetic" type and later became "nodular." The disease was rapidly progressive, and he died in 1915.

No records of the case are available, but photographs taken prior to 1907 invariably show his left hand in a sling, and he is said to have had a left ulnar neuritis. By repute he was very unhygienic, even dirty in his habits. It is recorded that he had tea and sandwiches served to him in the mortuary, and that he consumed these without washing his hands. In 1940 an old Bantu servant of the institution stated that he remembered

the doctor injecting ten members of the Bantu staff with suspensions of nodules to prove his point that leprosy could not be transmitted. Incidentally, none of them developed leprosy, so far as can be ascertained. I suggest that the doctor probably also injected himself, and concealed the fact that he had leprosy until after his return to Britain. He would have been segregated had he remained in this country. This is a certain instance of an adult over 50 years of age, from a nonendemic area, developing leprosy.

The second doctor (D.L.) worked here as a medical officer from 1919 to 1930. In 1929, while operating on a lepromatous patient, he pricked his left index finger. About three months later, at the age of 60, he developed a tuberculoid lesion at the site. Secondary lesions appeared and later resolved. He died 22 years later. His own report of his case, *verbatim* except for punctuation, reads:

I am unable to state accurately the date I pricked my finger when operating on an active nodular leper. It happened when I was stitching up the skin after removing a toe. As far as I can remember it must have been during the beginning of 1929, either January or March. I suffered no immediate discomfort or inflammation at the time, but some months later, about May or so, I noticed a slight swelling on the second front of 1st finger over the spot where I pricked the finger. Skin was only painful on pressure. In June I went to Robben Island where it gradually got worse, but not sufficiently really to worry me. My health was good, but I had a gone-in sort of feeling.

On my return to Pretoria I started taking smears. I found it hurt my fore-finger to hold the slides, and I had to use the middle finger and thumb. The thickening round the spot gradually enlarged and became more sensitive, and I began to get suspicions that it may develop into leprosy. I applied Tr. of Iodine on and off. On the 30th November I did an amputation on a kaffir. It was rather a difficult job due to adhesions and fibrosis. After I had finished both my hands felt numb, and it was quite a time before the feeling returned to normal. A few days later neuritis started in both my arms, with sharp shooting pains. My infected finger became red and inflamed, the redness starting from the pricked spot and gradually spread like a macule. My whole hand became swollen and raised red patches came out, one at the base of the first finger and another at the base of the thumb. At about the same time I had severe itching on the outside of my right ankle and then a typical macule gradually developed.

During Xmas and New Year, while these signs and symptoms were gradually developing, I felt very ill and the pains in my arms worried me a great deal. I had Diathermy and Xray treatment but this did not seem to do me much good, so I decided to have the fore-finger and the two raised macules removed, which Dr. Scholz was kind enough to do.

Strangely enough the removal of the finger did not release the pain in the finger and I am even now (June) aware of the finger. The swelling of the hand started going down soon after the amputation. Atrophy and superficial anaesthesia are gradually increasing and there is still a feeling of pins of needles on deep pressure on the arm and hand. The maculae on my ankle is also slowly increasing in spite of Xray treatments. I am getting chaulmoogra oil and creosote injections, my health has improved, and the pains are much less. My hand is still painful when I try to grip anything and is practically useless. The tips of the 2nd and 3rd fingers of right hand are hypersensitive and the peroneal nerve from the mac. to near the knee is also getting anaesthetic.

We have records of three European overseers in leprosy institutions here who contracted leprosy. The first (G. R.), was a South African and worked at Westfort from 1909 to 1915. He handled patients' clothing during his work. He developed tuberculoid leprosy in 1915, at the age of 32, and was discharged in 1922.

The second (R. H.), was born in England and came to this country as a soldier. He served as overseer at the Mjanyana Leper Institution from 1907 until he was pensioned in 1934. He was in daily contact with patients and their effects. He developed leprosy in 1940, when aged 66, and died of lepromatous leprosy in 1947.

The third case was born in the Orange Free State, in 1898. He entered the Basutoland Leper Institution when 42 years of age, and remained there for seven years. He states he never knowingly handled patients or their effects. He left there in 1949, and the first signs of lepromatous leprosy developed in 1953, when he was 55 years old.

Two of these five persons were born and reached manhood in Britain, and none of the other three had records of leprosy relatives. All developed leprosy in late manhood, and were presumably infected when adult. (I have excluded another European from this series as he did have distant relatives who had leprosy.)

When discussing infections in the Bantu staff, I fully recognize that leprosy is endemic in this country, even though the incidence of 0.77 per thousand is low. I have gone into the family histories so far as they are available, and have excluded one patient who had leprosy relatives. This leaves ten Bantu cases to report.

1. CASE No. 3329. Started work here, aged 27, as a police boy in 1902. In a riot in 1914 he was bitten on the face and hands. He developed lepromatous leprosy in 1919, when aged 44.

2. CASE No. 1162. Started work here, aged 31, as a police boy in 1900. In a fight with the patients he was badly wounded. He developed tuberculoid leprosy in 1909, aged 40.

3. CASE No. 4163. Worked as wagon driver in this institution for a European patient who had the coal contract. The European had had lepromatous ulcers, but worked on the harness and fed his employees. Six years after leaving here, the Bantu developed lepromatous leprosy.

4. DANIEL. Started work as dispenser's assistant at the Mkambati Leper Institution, when aged 36, in 1920. Developed lepromatous leprosy in 1926, aged 42.

5. MNYANZELI. Worked on the farm at Mjanyana from 1936 to 1938. Developed tuberculoid leprosy in 1943.

6. CASE No. 10479. When aged 25, he started work at Westfort in the hospital kitchen. He worked there for eight years, but after leaving he constantly visited the institution. He developed lepromatous leprosy when aged 42, 15 years after ceasing to work here.

7. CASE No. 3026. Worked as police boy for 12 years (one record says 29 years) at the Amatikulu Leper Institution. Developed tuberculoid leprosy ten months after retiring in 1940, aged 61.

8. CASE No. 3922. Laborer at Amatikulu for three years before he developed tuberculoid leprosy in 1947, aged 30.

9. CASE No. 3031. Worked at Amatikulu as police guard for eight years until 1933. Developed neural type leprosy when aged 79 years, in 1940.

10. CASE No. 3586. The information is incomplete. The man simply states that he worked at Amatikulu as a police boy and was later admitted as a patient in 1939, aged 30. Neural type.

The average age at which leprosy developed, in the 8 cases for which that is known, was 46 years; two were quite elderly. The average age at which leprosy develops among Bantu males generally is 19 years. I think these figures are evidence that the cases under discussion were infected when adult, and presumably during the course of their work in the leprosy institutions. I therefore make a plea that all workers in such institutions—and by no means do I exclude doctors—should be hygienic in their habits after handling patients or their effects.

The other concept which I wish to attack, because I consider it dangerous, is that leprosy can be contracted only after long-continued, close contact. This concept is carried to such ridiculous lengths that in some parts of the world lepromatous patients with positive smears are allowed to go home on holiday. In one institution I know of they are allowed home for two months a year. I think it would be just as reasonable to allow patients from a diphtheria ward to spend each Sunday at home. The following case illustrates the danger of exposing a child to infection for a period of six weeks.

A male native child, Siqaqa, was born in the female location at the Mkambati Leper Institution, Pondoland, on October 14, 1928. The mother (Manzinane) was a patient in the institution, and the father (Mampala) was also a patient at the time, but was later discharged. The child was removed from its mother and placed in the institution crèche on November 30, 1928. Contact, therefore, was six weeks. The mother died in January 1929.

During December 1929 the child began to show symptoms suspicious of leprosy, and was kept under observation. Early in August 1930 macules developed on the buttocks and left shoulder. A bacteriological examination was negative, but the diagnosis of leprosy was confirmed. After removal from the mother, the child had not been exposed to infection in any way.

It is the practice now in all South African leprosy institutions to remove babies at birth, and I know of no other instance of a crèche child developing leprosy. I have been informed that in one institution in Central Africa the children are breast fed up to the age of 6 months. I think this practice is to be condemned.

SUMMARY

1. Leprosy can be acquired by the adult staff in leprosy institutions.
2. A plea is made for elementary hygienic practices by the staff.
3. Infected individuals should not come in contact with children.

ADDENDUM: We have recently admitted one of our European ex-nurses as a patient. She started work here in 1935 when aged 22, and left to get married in 1939. Her health while she worked here was good

except for attacks of tonsillitis. She knows of no accident or trauma while at work. In 1955, when aged 42, she developed tuberculoid leprosy, which responded rapidly to DDS and atabrin. She knows of no leprosy in her family, or of any contacts with leprosy since she worked here. This appears to be another instance of an adult acquiring leprosy.

ACKNOWLEDGEMENT

I have to thank the Secretary for Health, Union of South Africa, for permission to publish this article.

RESUMEN

Disiente el A. rotundamente de los conceptos de que la lepra se adquiere sólo o principalmente en la infancia, y de que los adultos no tienen que tomar precauciones contra la infección.

Dos funcionarios médicos de la leprosería de Pretoria contrajeron la enfermedad. Dícese que uno de ellos inoculó a 10 miembros del personal de raza bantú (y posiblemente a sí propio) para demostrar que la lepra no es transmisible. El otro aparentemente adquirió la infección fortuitamente mientras operaba, apareciendo una lesión tuberculoidea en un dedo a los pocos meses de habérselo pinchado; se ofrece el propio relato de este sujeto.

Tres mayordomos europeos y una enfermera que habían trabajado en establecimientos sudafricanos también manifestaron lepra, sucediendo otro tanto a 10 miembros del personal de raza bantú sin antecedentes averiguables de lepra en sus familias.

Para ilustrar el hecho de que no se necesita una exposición prolongada, relátase el caso de un lactante nacido a una madre leprosa que no había tenido más que 6 semanas de contacto antes de trasladarlo a una casacuna, pero que manifestó lesiones un año después.

Abógase por el mantenimiento de reglas higiénicas por los miembros del claustro y por la prohibición del contacto con niños.

REFERENCES

1. KLINGMÜLLER, V. Die Lepra. Handbuch der Haut und Geschlechtskrankheiten, Band 10, Teil 2. Berlin: Julius Springer, 1930, pp. 204-206.
2. ROGERS, L. and MUIR, E. Leprosy. Bristol; John Wright & Sons, Ltd., 3rd ed., 1946, p. 84.