

THREE NEW CASES OF LEPROSY IN NORWAY

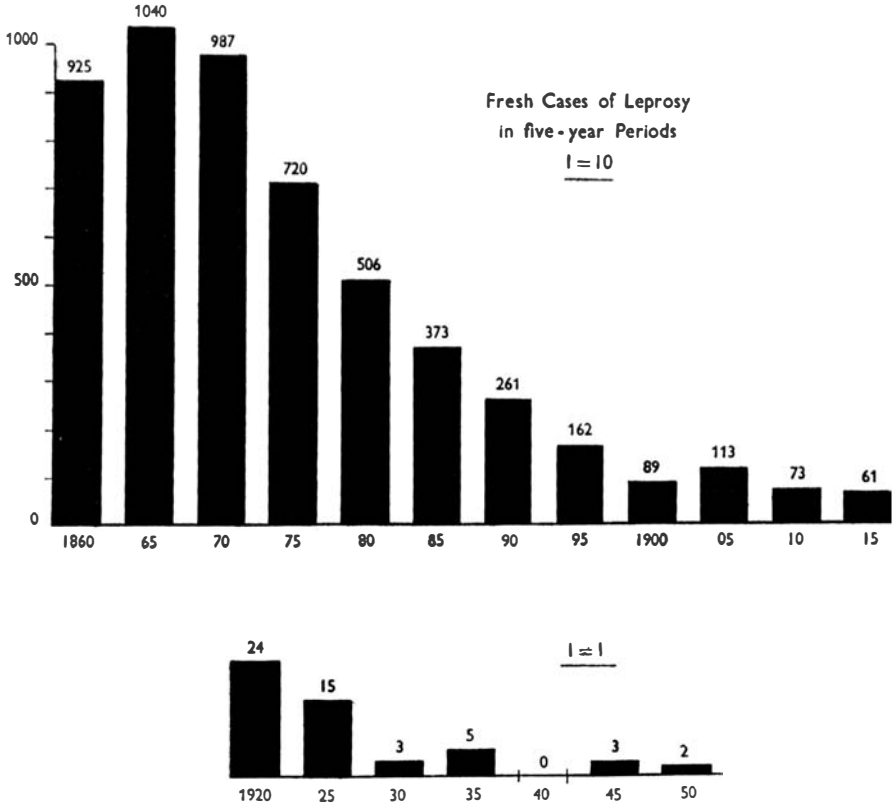
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Leprosy has existed in Norway from the oldest times. It is an open question whether this disease was introduced by the first human beings who colonised the country by immigration from the South and East thousands of years ago, or did not reach Norway till there was free communication with Western Europe in the bronze age and later. The disease evidently existed in Norway in pre-historic times, as witnessed by its mention in our oldest laws, and the variety of names given to it in old Norwegian. The disease increased greatly in Norway in connexion with the crusades as well as in Western Europe. It was during this period that our old leprosaria were founded. As in Western Europe, the disease regressed in the 16th and 17th centuries, but in contrast to what happened in western Europe, it increased again in the 18th and 19th centuries. The cause of this atypical behaviour of the disease is not known, but I am inclined to trace it to special conditions on the West coast of Norway, where housing conditions were worse than elsewhere in the country, and where there were great fisheries entailing close contacts of the fishermen during certain periods.

In the middle of the previous century, leprosy was endemic on the West coast of Norway from Lindesnes to Varanger, whereas it was practically unknown in the East of Norway inland, and on the South coast. It represented such a serious medical problem that radical measures had to be taken against it. Between 1850 and 1860, several new, large leprosaria were founded and one of the old leprosaria was enlarged and modernised by the public authorities. Further, the first law concerning health commissions was adopted, being applicable at first only to those districts in which leprosy existed, and referring only to this disease.

Owing to these measures and assuredly also because of a general improvement in hygiene, and perhaps most of all on account of better housing conditions, there has been a marked regression of the disease in the course of the last hundred years. In relation to the number of inhabitants, there are today far fewer cases of leprosy in Norway than in the U.S.A. for example, and in this respect we are just as well off as the rest of Western Europe with the exception of Denmark,—the only country in the world without a single case of leprosy. But even though the disease has regressed markedly, we still have a total of 11 cases, and from year to year

new cases continue to crop up. In the first place, we are not quite finished with the epidemic of the previous century. The disease runs a very chronic course and has a very long incubation period. Now and again cases appear which must be traced to our old infection. Since 1930, we have had 9 such cases. We also have a small number of new infections introduced by seamen returning home infected. Since 1930, we have had 3 such cases.



The disease is without doubt infectious, but the degree of infectiousness is very slight. Some leprologists have insisted that infection does not occur in adult life. This teaching is disproved, among other things, by the three above-mentioned seamen. On the other hand, the same state of affairs shows that the susceptibility of adults to the disease is very slight, for it is certain that hundreds of Norwegian seamen have been exposed in the past to quite massive infection (in bars and brothels in tropical harbours).

In less than one year we have observed 3 new cases of leprosy in sibilings,—2 men and a woman. There has been much leprosy in their family, and their mother as well as two grandmothers

suffered from the disease. The family is healthy. Of their 10 children, 3 died when young, and 4 are still alive and well.

In 1931 the mother was admitted to the leprosy hospital in Bergen suffering from quite advanced lepra tuberosa. She died in 1933 of a disease of the biliary tract. The following is a short account of these 3 new cases:

A man, S.M., born on October 9th, 1925, was admitted to the leprosy hospital on January 10th, 1951, presenting lesions of the skin and the peripheral nervous system. The skin of his face presented patches of a light brown colour without definite maculae. Owing to slight facial paralysis on the left side, he could not shut his left eye completely, and the left corner of his mouth was drawn somewhat up. The left side of his face presented definite hypoaesthesia of all the sensory qualities. There was no loss of cilia or of supercilia.

There was a maculo-papular rash on the trunk and limbs. Yellowish-brown patches, to some extent quite sharply defined, merged into larger, confluent areas which were scattered in an irregular fashion over his arms. These patches showed no definite infiltration and were not raised. There were several such patches on the trunk, both in front and behind. On the legs there were large, confluent patches of the same appearance and character as those already described. About 10 cm. above the inguinal region, these patches stood out sharply defined against the normal skin. On the anterior aspect of both thighs was an irregular, whitish area of scarring. Scattered over thighs and legs were numerous nodules up to the size of a pea, being sharply defined, firmly elastic, and of a brownish-red colour.

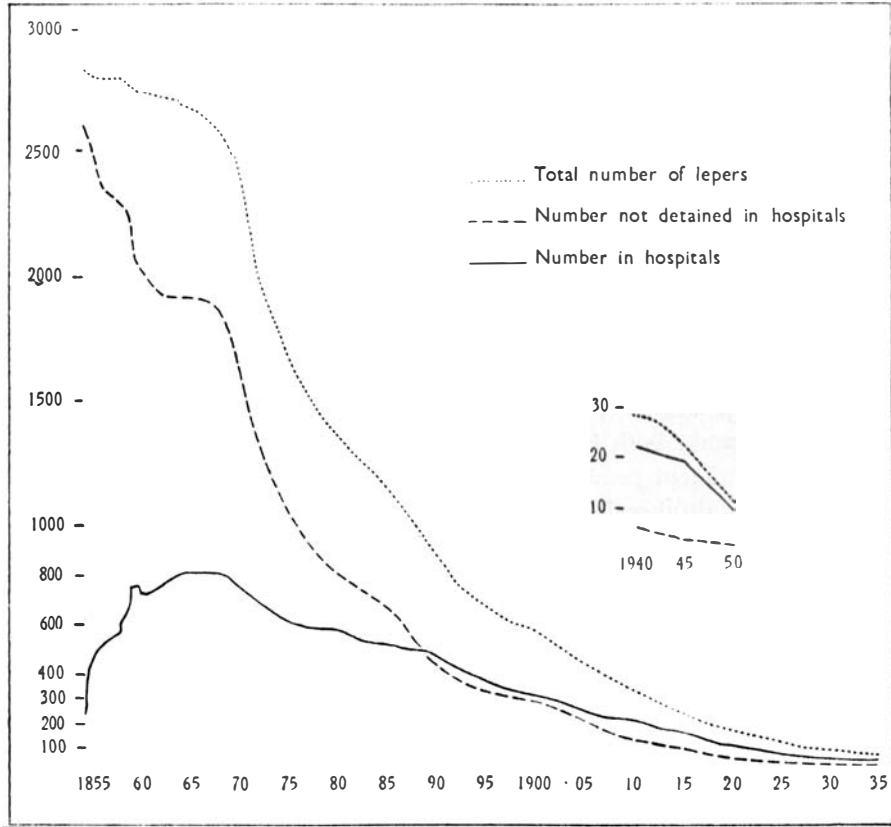
The appearance of his hands was very striking, with marked symmetrical atrophy of the muscles, most evident in the thenar and hypothenar areas. There was a slight flexion contracture of the fingers. There was marked hypoaesthesia for *all* the sensory qualities, from the fingers to the middle of the upper arms. His legs also presented definite sensory disturbances of the same character, from his toes to the middle of his thighs. The reduction of sensation in his skin did not seem to be more marked over the patches of rash than over the rest of his limbs. He said that skin sensation was practically intact in the patches on the trunk of his body.

Both the ulnar nerves were tender on pressure, as were also both peroneal nerves at the point where they appeared near the head of the fibula. Both forearms showed scars from burns.

Numerous leprosy bacilli were found in the secretion from his nose.

Biopsy of a nodule showed the typical picture of lepra tuberosa with numerous leprosy bacilli, both diffusely scattered and in typical globules.

A woman, M.M., born on September 28th, 1923, was admitted to the leprosy hospital on January 3rd, 1951. She was a couple of years older than her brother, and she presented a milder degree



of the same clinical picture. She also suffered from marked, symmetrical atrophy of the muscles of her hands and a slight flexion contracture of her fingers with loss of sensation almost to her shoulders. Her big toe was thickened and discoloured a bluish-red. Under the ball of her big toe was a scar from a perforating ulcer. Loss of sensation extended up to the middle of both feet. The left ulnar nerve was thickened and tender on palpation. She, too, presented scars from burns of her forearms. On the extensor aspect of her upper arms were several small cutaneous and subcutaneous brown nodules. One of them was larger than the others and was

more reddish-brown in colour. The typical picture of a leproma was found on biopsy. On the dorsum of the right foot was a small, diffusely limited infiltration with a brown discolouration. On her left leg there was a small bluish-red infiltration. Apart from these findings there was no rash.

A few leprosy bacilli were found in the secretion from her nose.

It was easy to diagnose leprosy in both these cases as soon as this possibility occurred to us. The symmetrical involvement of both motor and sensory peripheral nerves in association with demonstrable thickening and tenderness of peripheral nerve trunks is pathognomonic of leprosy. The young man's maculo-papular rash was also very characteristic of this disease. The only possible alternative diagnosis was mycosis fungoides, but its histology is quite different. Whenever possible, the diagnosis of leprosy should always be verified by the demonstration of leprosy bacilli.

A man, G.M., born on January 18th, 1913, was admitted to the leprosy hospital on September 1st, 1951, suffering from a papular rash on trunk and limbs. Numerous nodules, some cutaneous and others subcutaneous, ranged from the size of a pin's head to that of a pea. The cutaneous nodules were sharply defined, firmly elastic, and of a brownish-red colour. The subcutaneous nodules were more diffusely limited and presented a bluish-red colour. A typical leproma was found on biopsy.

There was a suspicion of a slight bilateral atrophy of the thenar area without any muscle atrophy elsewhere. His hands, forearms and feet up to the knees showed definite loss of sensation which was most marked for cold and heat, less so for touch and not existing for pain. Both forearms showed scarring after several burns.

This patient was at sea when the disease was discovered in his brother and sister. I notified his ship's doctor who let several leprologists in the Pacific examine him. None however made a definite diagnosis.

The behaviour of the disease showed many features in common in these three patients, all of whom must have been infected by their mother more than 20 years ago. The first patient, S.M., noticed in 1940 that his hands had become remarkably thin. He consulted a nerve specialist who diagnosed a peripheral nerve lesion and prescribed electrical treatment. In the summer of 1950 the rash began to appear, but he did not attach much importance to it.

The second patient, M.M., stated that her hands had been thin as long as she could remember. In 1949 she developed, under the ball of her left big toe, a perforating ulcer which was excised by a surgeon with transplantation of skin. When, however, the ulcer relapsed in the autumn of 1950, she was referred to me.

The third patient, G.M., noticed that during the post-war years he was liable to develop sores from burns on his forearms. He believed that the rash began to develop during the spring of 1951.

In these three cases it is quite certain that the disease must have begun with involvement of the peripheral nerves, and a peripheral nerve lesion was the diagnosis in 1940 of a neurologist in the case of the youngest patient, S.M. All three patients presented several scars, some of them quite large, on their forearms after burns. The four healthy siblings did not show this condition. In all three cases a tuberous leprosy had developed in the course of 1950-51, 20 years after the last possibility of infection.

These case records illustrate well, among other things, the markedly chronic course run by the disease. There is no other disease with so long an incubation period and protracted course. As already pointed out, 4 of the 7 surviving siblings are healthy. One of the healthy siblings and one of the infected ones were non-identical twins. The father is healthy. This family tragedy shows that the infection usually occurs in childhood even when the disease becomes manifest in adult life. A prolonged and massive exposure is necessary for infection to occur, and even under such conditions not one half of the siblings in this particular family developed leprosy.

It seems natural to assume that the development of leprosy depends on:

- (1) Massive exposure to infection,
- (2) Great reduction of resistance at the same time as, or directly after, such exposure while the bacteria are vegetating in the organism.

Both these two conditions probably existed in this family, the children being assuredly exposed to infection for several years, during which they had had periods of much reduced resistance due to intercurrent infectious disease or poor nutrition.

As a supplement to these three cases, I would like quite briefly to record a fourth case which presented considerable difficulties with regard to differential diagnosis:

A man, aged 77, came from the north of Norway (Nordland), and was admitted to the leprosy hospital in the summer of 1945. He did not know of any case of leprosy in his family or in the neighbourhood. Some 5-6 years before admission to hospital he had noticed loss of sensation in his hands. Flexion contractures of his fingers developed gradually, and he was treated in hospital on several occasions for persistent sores on his hands. In the summer of 1944 he was admitted to the Riks-hospital under the suspicion of leprosy. On examination at the Skin and Neurological

Departments, the choice of diagnosis between leprosy and syringomyelia was left undecided, though the latter diagnosis was regarded as the most likely.

When I examined the patient in the spring of 1945, I observed a definite symmetrical atrophy of the muscles of his hands and loss of sensation for all the sensory qualities over both his hands to the middle of his forearms. He also presented a peroneus palsy on the right side and bilateral loss of sensation from his toes to a point half way up his legs below the knees. On the clinical evidence I diagnosed a typical maculo-anæsthetic leprosy. In the hope of verifying this diagnosis, I secured the excision of a small section of his left ulner nerve. I had hoped to find histological evidence of scarring which would do much to confirm the diagnosis of leprosy. What I did find were typical leprosy bacilli.

I have reported these cases to recall to mind that cases of leprosy can still crop up in Norway. Medical officers of health in particular, with archives giving information about families in which leprosy has occurred earlier, should keep this possibility in mind. Further, we import the disease occasionally with seamen who have become infected when sailing in the tropics. The diagnosis of leprosy should be kept in mind when we are confronted by obscure cases of disease of the skin and nervous system.