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Leprosy in Malaysia

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Abstract

Leprosy is a chronic infectious disease and is still a public health problem in Malaysia. In 1926, the Leper Enactment Act was established which required compulsory notification and isolation of leprosy patients. As a result, the National Leprosy Control Centre (NLCC) was built in Sungai Buloh, Selangor. In 1969, the National Leprosy Control programme was launched with the objective of early case finding and decentralisation of treatment of leprosy. The treatment of leprosy patients is integrated with basic Medical and Health services in Malaysia. With the implementation of multiple drug therapy in 1985, the National prevalence rate of leprosy has reduced from 5.7 per 10,000 in 1983 to 1.7 per 10,000 in 1992.

The Research Unit in NLCC was established in 1950, where cultivation of *Mycohacterium leprae* using mouse foot-pad technique is done. This technique is used for assessment of efficacy of chemotherapeutic agents in leprosy. Research activites are also done in collaboration with the Institute for Medical Research in Kuala Lumpur such as isolation of *Mycobacterium leprae* antigen using T cell clones and phenolic glycolipid antigen.

Key words: Leprosy, mycobacterium leprae, epidemiology.

INTRODUCTION

Although the incidence of leprosy in Malaysia is declining, it is still a public health problem. The disease was probably introduced into the country in the nineteenth century by Chinese and Indian immigrants among whom the disease was prevalant. In 1926 the Leper Enactment Act was enforced, requiring compulsory notification of all leprosy patients and mandatory isolation and hospitalisation. The National Leprosy Control Centre (NLCC) was built in Sungai Buloh, Selangor in accordance with the Act. The centre covers an area of 562 acres of low hilly land and is among the largest in the British Commonwealth. It has a hospital with 855 beds, an old leprosy settlement of 2000 bed chalets, an administration block and a research unit.

The National Leprosy Control programme

The National Leprosy Control programme was launched in 1969 in West Malaysia and extended into the East Malaysian states of Sarawak and Sabah in 1974 and 1985 respectively. The main objective of the programme is to control leprosy by early case finding and effective treatment, decentralisation of the treatment of leprosy and the abolishment of automatic entry for permanent settlement in the NLCC. The diagnosis of leprosy is confirmed by skin or nerve biopsy. The treatment of leprosy patients is fully integrated with basic medical and health services in Malaysia. This has resulted in 63% decline in the number of patients in the NLCC over a span of 20 years (1970-1990).[1]

Treatment

The sulphone era for general treatment of leprosy began in NLCC in 1948. The National Research unit in leprosy was established in NLCC in 1950. The main activity of the unit is the assessment of the efficacy of chemotherapeutic agents. In 1964, the first sulphone-resistant cases of leprosy were detected in Malaysia. This led to the usage of other drugs. WHO multiple drug therapy (MDT) was implemented in 1985. The National Leprosy Control Centre has modified the regime and this modified regime is used in the country. The drugs used in MDT are rifampicin, clofazimine and dapsone. The duration of the treatment depends on the clinical type of leprosy. Paucibacillary type of leprosy is treated up to 1 year and in the multibacillary type, therapy is continued for 3 years or up till the smears are negative for bacilli. Patients are hospitalised only during the intensive phase of the treatment and continue to take medication at home during the maintainance period. The advantages of MDT include shorter duration of treatment,

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decrease in deformity rate and low defaulter rate. However, the patient compliance is still a problem. Hyperpigmentation of the skin due to clofazimine is most unacceptable to light skinned patients. With implementation of MDT the National prevalence rate of leprosy has reduced from 5.7 per 10,000 in 1983 to 1.7 per 10,000 in 1992. Records from the National Registry of Leprosy in 1992 showed the total number of cases in the country was 2989. The new cases detected were 293 for 1992.[2]

Role of Pathology in leprosy

Microscopical examination of the skin or nerve lesion plays a major role in the diagnosis of leprosy. A typical clinical lesion should be biopsied. Routine stains used in the histological study are haematoxylin and eosin (H&E) and the modified Fite stain for acid-fast bacilli (AFB). The Ridley-Jopling classification' is the most widely used histological classification. The histological spectrum of classification consists of full tuberculoid (TT), borderline-tuberculoid (BT), mid-borderline (BB), borderline-lepromatous (BL) and lepromatous (LL). The microscopical diagnosis of multibacillary leprosy such as BL or LL type is easy due to the presence of numerous AFB. The diagnosis of early leprosy such as indeterminate and tuberculoid leprosy is often difficult clinically as well as histologically. Serial sections of the biopsied tissue may be necessary to detect granulomas around nerve bundles. Immunocytochemical staining using S-100 protein as a nerve marker has been attempted in leprosy.[4] It is a valuable marker to detect the nerve fibres. Fragmented and dissociated nerve fibres in the granuloma of tuberculoid leprosy may not be easily seen in routine H&E sections. S-100 staining is useful to pathologists who only occasionally get biopsies from leprosy patients.

Reactions and relapses pose problems in the management of leprosy. Reactions in leprosy include erythema nodosum leprosum (ENL), exacerbation reactions, Lucio reaction which usually occur in LL type of leprosy and delayed hypersensitivity reaction occuring mainly in the BL-BT range of leprosy. Clinical evaluation of reactions and relapses is often difficult because both conditions present with signs of acute inflammation.[5] In such situations, biopsy of the lesion is useful. Histology of the typical ENL lesion shows infiltration of the granuloma by polymorphonuclear leucocyte and AFB are usually scanty and degenerated. In exacerbation reaction, there is a polymorphonuclear leucocyte infiltrate, but the bacterial load is exceptional and majority of the bacilli are solid staining forms.

During the 5-year period between 1981 to 1985 autopsies were performed in 35 leprosy patients in the University Hospital, Kuala Lumpur.[6] These subjects were unclaimed bodies of inmates of the NLCC. The patients were elderly subjects with a mean age of 74 years. Thirty patients were Chinese and five were Indians. The clinical duration of the disease varied from 10 to 30 years. Twenty three (66%) patients had lepromatous leprosy. Infection was the most important cause contributing to death (40%). Twelve patients (34%) had pyogenic bronchopneumonia. Pulmonary tuberculosis was noted in 10 (28%) cases and in two of these cases, there was tuberculous bronchopneumonia. The other causes of death were cardiac failure (20%), renal failure (17%), malignancies (11%), intracerebral haemorrhage (6%) and perforated peptic ulcer (6%). Renal changes were seen in 25 (71%) of patients and contributed to the high morbidity. The renal changes may be caused by drug reaction, related to prolonged exposure to sulfones, or due to altered immunity with increased susceptibility to infection. Generalised amyloidosis was observed in 5 (21.7%) of 23 patients with lepromatous leprosy and 1 (8.3%) of 12 patients with tuberculoid leprosy. The major organs involved by amyloidosis were the kidney (100%), heart (100%), adrenals (100%), liver (83%), spleen (83%) and lungs (83%). Amyloid deposition was observed mainly in the walls of medium-sized arteries. None of the cases showed amyloid deposition in the brain.

Research in leprosy

The mouse foot-pad technique for cultivation of *Mycobacterium leprae* was started in the Research Unit of NLCC, Sungai Buloh in the year 1969. Presently this technique is used for drug susceptibility testing and the drugs tested include dapsone, clofazimine and rifampicin. A study done between 1985-1990 showed a high percentage of dapsone resistance (22.4%).[7] Within this group, dapsone resistantance was primary in 20% of cases and secondary in 71%. Patients with secondary dapsone resistance had either previous monotherapy or a history of relapse.

The isolation of M. *leprae* using T cell clones and Phenolic Glycolipid antigen (PGL) is being carried out in the Institute of Medical Research

(IMR) in Kuala Lumpur. A three-year National seroepidemiological study of PGL involving about 40000 individuals selected by stratified sampling was carried out.[8] The study showed that the seropositivity of PGL antigen was correlated with the prevalence rate of leprosy in a defined population. High antibody titres of IgM class were found in higher frequencies among individuals residing in high prevalence communities and in leprosy patients. IgG antibodies were found in significant quantities in individuals from areas of low prevalence and in leprosy patients treated over a long duration of time.

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