Leprosy in New Zealand
Stephen T. Chambers

The article published in this edition of the Journal serves as a welcome reminder that leprosy is not only of historic interest in New Zealand, but a persisting clinical challenge. From 2004 to 2013 there were 38 cases of confirmed or probable leprosy, all of which were imported. The major countries of origin were from the Pacific region, particularly Samoa and Kiribati, although there were cases from further afield, including the Philippines, India and Ethiopia. Recent figures from WHO (2012) demonstrate that there are about 250,000 cases reported each year, and three-quarters of these are in the Asian region, with Brazil and Nigeria contributing most cases from outside Asia. Unfortunately, the number of cases has remained at about this level over the past 10 years, after a rapid decline following the widespread adoption of multidrug therapy (MDT) in 1990s. Given the changing migration patterns into New Zealand, more cases can be expected in migrants from endemic countries.

The epidemiology is slowly evolving. In 1991, WHO set a goal of eliminating leprosy as a public health problem by the year 2000. The target was that there should be a prevalence of less than one per 10,000 of the global population, and individual countries were encouraged to ensure this target was met. This proved to be an achievable goal and has led to the control of leprosy by this criterion. However, the reduction in numbers has meant that specialist leprosy programmes at the public health level are not sustainable and leprosy services have, by necessity, become integrated into the mainstream health systems. This raises the conundrum of how awareness, diagnostic and therapeutic skills for leprosy can be maintained. Where skills are lost a resurgence in cases may pass unnoticed for some time, as the incubation period is commonly 3–5 years, but may be as long as 20 years, and it has an insidious clinical onset. Governments and health providers find it difficult to provide an ongoing focus on a rare disease when there are epidemics of more pressing immediate concern—such as obesity, diabetes and heart disease. This led WHO to classify leprosy as a neglected tropical disease.

In New Zealand the main clinical issues are of early recognition, and the skills to treat the severe reactions often associated with treating leprosy. The cardinal clinical features of leprosy are non-itchy hypopigmented, erythematous or infiltrative lesion, with or without neurological signs or symptoms and peripheral nerve thickening. The clinical manifestations usually begin in the indeterminate phase with a single, or a few, ill-defined hypopigmented or faintly erythematous patches that are easily overlooked. They then may develop into paucibacillary disease (up to five lesions), lepromatous disease or persist in an indeterminate form of leprosy, depending on the immunological response of the host. It can thus present as a bewildering array of non-itchy lesions, ranging from a solitary hazy macule to inflamed large patches, scores of shiny nodules, or diffuse infiltration of cooler skin areas in the face and ears. It is difficult to believe that such varied and divergent manifestations are caused by the same organism. While the first signs are usually in the skin, and invasion of the peripheral nerves follows, occasionally neural disease is the first manifestation with enlarged painful peripheral nerves that may be accompanied by weakness, loss of sensation and sweating. The diagnosis can be confirmed by identification of Mycobacterium leprae in skin biopsies, split skin smears and PCR testing.
Management of severe reactions following treatment are common and challenging. Type 1 reactions usually occur in borderline leprosy. The existing lesions show signs of acute inflammation following a change in cell-mediated immunity and nerve swelling and paralysis may suddenly develop, causing long term disability. Erythema nodosum leprosum or type 2 reactions tend to occur late in the course of lepromatous disease and are not located within existing lesions, but elsewhere on the body. These lesions may come in crops and appear as nodules or plaques and become vesicular, pustular or gangrenous and break down. Fever and malaise are common accompaniments. Nerve damage is less common, but joint pain, iritis, iridocyclitis, tibial pain and epididymo-orchitis may develop. The cornerstone of treatment is steroid therapy but clofazimine has an important role, as it has immune-modulatory effects aside from its antileprotic effects. Fortunately international expertise is available for consultation in difficult cases through the Pacific Leprosy Foundation, as well as other consultative services.

In Pacific countries such as Kiribati, Federated States of Micronesia, Samoa, Solomon Islands and Papua New Guinea, there are not only limited diagnostic and treatment resources but social conditions favour spread. For example, crowding is a major problem in Kiribati for example, where 100,000 people live on an area of 726 square kilometres in extremely poor housing conditions. Spread is thought to be primarily from the nasal mucosa as untreated patients can produce ten organisms daily, which persist in the air for many hours. Infection is thought to be via inhalation and entry through the nasal mucosa and possibly through abraded skin. In addition, the stigma associated with leprosy may significantly reduce job prospects, family esteem, marriage opportunities and social interactions. This restricts the willingness of patients to seek help early and increases the risk of spread.
EDITIORIAL

REFERENCES:
1. Reference paper (JB NOTE)

URL: