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Short Communication

Neurological Manifestations and Brain Disorders of Leprosy

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Introduction

Leprosy (also known as Hansen's disease) is an important global health concern caused by Mycobacterium leprae and Mycobacterium lepromatosis [1,2]. Clinical manifestations range from cutaneous manifestations to neurologic disability and blindness [3]. Early diagnosis and treatment are important for minimizing the neurologic disability.

Epidemiology

The prevalence of leprosy is variable. Leprosy has been reported in Canada, Asia, Mexico, South America and Central America [4]. The greatest number of new cases were seen in Brazil, India, Bangladesh, Indonesia and Nigeria. However, with increasing international travel, patients with leprosy can be seen anywhere [5]. In general, the disease is more common among males with a ratio of 1.5: 1 [6].

Transmission and risk factors

The disease is spread by nasal discharge and respiratory route, even though the means of transmission are not fully understood**.** Handling, killing or eating armadillos have been reported as a transmission pathway in some cases. Infection can develop due to many factors such as genetic factors and immunological status [7,8]. Close contact, leprosy type in index patients, armadillo exposure, age, genetic factors (mainly PARK2/PARRCG genes) and immunosuppression may be considered as major risk factors [9,10].

Classification and terminology

- **Ridley-Jopling classification**: This classification is primarily based on the findings of a cutaneous, neurological and biopsy correlation with immunologic response. Leprosy is classified using the following categories: tuberculoid (TT), borderline tuberculoid (BT), mid-borderline (BB), borderline lepromatous (BL), lepromatous (LL), and indeterminate (I) [11].
- **WHO classification**: This classification is based on the number of skin lesions present. According to this system, patients with five or fewer skin lesions without detectable bacilli on skin smears are classified as 'Paucibacillary leprosy (PB)' and those with six or more lesions as 'Multibacillary leprosy (MB)' [12].

Clinical manifestations and diagnosis

Physical exam findings include hypopigmented skin patches, loss of sensation in involved areas, painless wounds, paresthesia and enlarged peripheral nerves. Neuropathy and ophthalmic injury can also be seen. As immunologic reactions, systemic inflammatory complications can occur before or during treatment. There are two types of leprosy reactions: type 1 and type 2. Type 1 occurs in patients with borderline disease and type 2 with lepromatous disease. The diagnosis is established when at least one of these findings is present and skin biopsy confirms the presence of acid-fast bacilli in a cutaneous nerve [13,14].

Neurological manifestations in leprosy

Hansen's disease, or leprosy, was first identified in 1873 by Gerhard Armauer Hansen. Mycobacterium leprae, an acid-fast bacterium causes the disease and primarily infects Schwann cells in the peripheral nervous system. The incubation period is seven years on average and ranges from three months to 40 years [15]. The neurologic manifestations of leprosy occur in the central nervous system, peripheral system and also the spinal cord [16]. At the time of diagnosis, approximately 60% of patients have evidence of peripheral nerve damage [3]. As a result of neural tropism of leprosy bacillus, Schwann cells are affected and eventually lead to demyelination [17].

Sensitive electrophysiological methods have demonstrated that peripheral nerve damage occurs earlier in lepromatous disease than tuberculoid disease [18]. Even after antibiotic treatment,

many dead bacterial cells remain silent within the nervous system and produce immunological response as acute or chronic neuritis [17]. The optimal growth occurs at 27-30°C. This is why it tends to settle in superficial and cooler areas, such as the skin, nerves, testes and upper respiratory tract [19].

Peripheral neuropathy is more common in patients with sensory defects, but painful neuropathy can also occur in later stages of the disease. In tuberculoid disease, sensory and motor loss generally occurs in the distribution of nerves and skin lesions, while nerve damage is more generalized in lepromatous disease. Nerve trunks including ulnar and median nerves, the common peroneal nerve, the posterior tibial nerve, the facial nerve, the radial cutaneous nerve and great auricular nerve are commonly affected [20,21].

In detailed physical examination, hypopigmented skin areas and sensitivity in the affected nerve trace can be observed. 4-8% of patients have no dermatological involvement and are known as 'pure neural form' or 'pure neuritic leprosy (PNL)'. In these patients, clinical diagnosis needs histological confirmation by nerve biopsy. The dorsal ulnar cutaneous, the medial/lateral antebrachial, or the superficial radial or sural nerves are the most suitable nerves for biopsy. Paresthesia is the most common manifestation (55%) in PNL, followed by motor dysfunction (24%), neural tenderness (12%) and sensory loss (8%). In clinical evolution, the most common presentation is mononeuritis multiplex, followed by mononeuropathy and polyneuropathy [16].

In leprosy, 6.9% of neuritis occur without other signs of reactions and are known as 'acute neuritis'. Sensory and motor dysfunction without skin signs or any positive symptoms are called 'silent nerve paralysis' and occur in 7% of newly presenting patients. Due to the involvement of small unmyelinated fibers, autonomic dysfunction is also commonly seen and can lead to dry skin and cardiac and respiratory dysautonomia. In 10-17% of patients, cranial nerve involvement can be observed. The facial and trigeminal nerves are the most frequently involved and can cause lagophthalmos, corneal xerosis, anesthesia and facial muscle weakness. Superficial nerve enlargement can be a clue in early diagnosis. The vestibulocochlear, the glossopharyngeal, the vagus and the hypoglossal nerves are less commonly involved [16,22].

Early diagnosis and treatment of neuropathy is important in reducing peripheral nerve damage and controlling the disease. We need to expand our knowledge of predisposing factors and triggering mechanisms [3]. Multidrug therapy and steroids are the most common medical treatments. In some cases, surgical decompression can also be used as an adjunctive treatment [23].

In summary, leprosy cause significant neurological morbidity worldwide. It is important to be aware of the common neurological manifestations of leprosy for early diagnosis to prevent and minimize injury to peripheral nerves [22].

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