Nerve damage, surgery and rehabilitation in leprosy

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Introduction

Leprosy is the only bacterial disease characterized by the causative organisms infecting the peripheral nerves. Clinical or histological demonstration of peripheral nerve involvement in the skin lesion or elsewhere is an essential diagnostic criterion for leprosy. The consequences of peripheral nerve involvement result in great morbidity and social stigma associated with leprosy. Notwithstanding all this, our knowledge of nerve involvement and damage in leprosy is sketchy and our ability to modify the course of events limited.

Stages of involvement

Five stages of nerve involvement can be recognized, the first two being identifiable only by histological scrutiny while the later three are clinical.

(i) Stage of parasitization: This is probably the earliest stage but can also be the transition phase between "disease" and "non-disease" states since some contacts of patients may also exhibit this phenomenon (Khanolkar, 1951). In this stage M. leprae are found inside the nerve, usually in the Schwann cells, and there is a yet hardly any host tissue response. The issue of "affinity" between Schwann cells and M. leprae still remains unsettled.

(ii) Stage of tissue response: Persistence and probably multiplication of the bacilli eventually evokes a tissue response which is initially non-specific or indeterminate but, becoming specified later as tuberculoid, borderline or lepromatous granuloma as the immunologic status becomes better defined (Iyer, 1965). It is possible that this sequence is neither necessary nor invariable. Neural granuloma cells do not appear to be qualitatively different from those of the dermal granuloma (Narayanan, 1988). In some patients, however, the neural lesion has been found to be immunologically more deficient than that of the skin lesion (Srinivasan et al., 1982a).

(iii) Stage of clinical infection: As the tissue response increases the nerve gets thickened and becomes clinically recognizable as such. There is no noticeable neural functional deficit although some positive sensory phenomena (formication, tingling, numbness etc.) may be present. One cannot predict the eventual clinical outcome from the signs and symptoms even at this stage.

(iv) Stage of nerve damage: Eventually neural functional deficit becomes clinically demonstrable when sufficient number of nerve fibres become damaged. Initially the unmyelinated and thinly myelinated fibre-mediated functions (thermal sensibility, pain perception, sweating) are affected, followed in course of time by loss of the thickly myelinated fibre-mediated functions. Loss of sensibility occurs earlier than motor paresis or paralysis. Nerve damage is reversible at this stage.

(v) Stage of nerve destruction: This is the end stage of nerve involvement in leprosy. Conducting elements are totally destroyed and collagenized (Iyer, 1965). Caseation and cold abscess formation may occur in tuberculoid leprosy.

The five stages mentioned above are neither universal nor invariable. The time and duration of each stage may vary from person to person, nerve to nerve and even in the different parts of one nerve, indicating the operation of local risk factors or, even possibility, different modes or times of infection.

The causative factors involved in the above scenario seem to have a network rather than a linear relationship. They include M. leprae (antigens/products), immunologic and non-immunologic processes, inflammation, internal and external compression and possible vascular mechanisms (Carayon, 1985). Long term use of a potentially neurotoxic drug like dapsone may also be contributing to nerve damage (Srinivasan and Nordeen, 1966; Gupte, 1979; Radhakrishna and Nair, 1987).
Location and patterns of nerve involvement, damage and recovery

Dermal nerve twigs, cutaneous nerves and major nerve trunks are all affected in leprosy, but, from the point of view of morbidity, involvement of major nerve trunks is the most important. It is mainly the nerve trunks of the extremities that are thus affected. Some of them are involved more often (e.g., ulnar, posterior tibial) and some fully recover more often (facial, radial) than the others. The reasons for such variation in behaviour are not clearly understood.

Onset and progress of nerve damage

Clinically, nerve damage may occur insidiously, or, episodically (associated with attacks of acute or subacute neuritis), or, catastrophically as a single irreversible event. Insidious onset and gradual progress appears to be the most common mode and is known as "quiet nerve paralysis" (Srinivasan et al., 1982b).

Consequences of nerve damage

The consequences of nerve damage extend far beyond the nerve and the structures it supplies. These are best appreciated by applying the three tier (impairment-disability-handicap) model developed by rehabilitation scientists (WHO, 1980; Srinivasan, 1984).

Impairments refer to the anatomical, physiological and psychological abnormalities resulting from the disorder. These primary impairments may, in turn, give rise to secondary abnormalities or secondary impairments. The common primary impairments seen in leprosy are nerve involvement and damage, damage to facial structures, ocular involvement and damage and personality disorders, of which the first is the most common. In most cases, these primary impairments are reversible to begin with, but become permanent later on. The secondary impairments are mostly the consequences of unprotected use of insensitive extremities and are collectively known as "anaesthetic deformities" and include ulceration of feet and hands, shortening and contractures of digits and skeletal disorganization usually of the tarsus.

Disabilities result from impairments which render the person unable to carry out certain acts considered normal. This inability is a disability and leprosy patients may suffer from disabilities involving manual dexterity, locomotion, sight, personal care, communication and behaviour.

Handicaps are the social disadvantages experienced by disabled persons. Leprosy patients often experience handicaps relating to social status, occupation, economic independence, mobility and physical independence. Persistent handicaps loosen societal bonds. The affected persons are relegated to the fringe of the family and society and are eventually pushed into "colonies" and unsavoury occupations causing their "dehabilitation". Finally, the affected person may become totally alienated from all society and end up as a destitute.

Corrective surgery

Corrective surgery is a part of this strategy primarily aimed at preventing handicaps, by making disabled patients able again. This branch started developing forty years ago and significant progress has been made over these years. During the earlier years the stress was on developing corrective procedures. Subsequently, efforts were spent on understanding the defragments and, more recently, the emphasis has shifted to developing interventions to prevent disabilities and deformities. Today we perceive surgery and allied measures as having two closely related objectives:

1) prevent handicaps by making patients able and normal looking once again and
2) prevent the onset and worsening of disabilities.

Hands: Stigmatizing deformity and loss of manual dexterity because of loss of sensibility and muscle paralysis are the main problems in hand. Time-tested surgical procedures are now available for correction of all the standard paralytic deformities of the hand and also deformities of the face (Brand, 1959; Fritschi, 1971; Antia and Dover, 1978; Srinivasan, 1978a).
results of these procedures have been highly satisfactory. The disability of loss of sensibility in hands can be overcome to a considerable extent by sensory re-education by which the patient learns to utilize the residual sensibility in the hand and re-interpret the sensations meaningfully (Dellon, 1981). We may expect this technique to be increasingly exploited to help leprosy patients in the coming years.

Feet: The major problem here is progressive destruction by repeated ulceration due to walking and the resulting periodic disability of inability to get around. The other, lesser, problem is drop-foot which makes walking ugly and difficult and also renders the foot vulnerable to ulceration and destruction. Our objective is to restore the ability to walk without endangering the feet. While uncomplicated ulcers are easily healed by simple dressing, protective foot-wear and limiting walking, orthopaedic and plastic surgical procedures are available now for even the complicated ulcers (Srinivasan, 1976; Srinivasan, 1982). Drop-foot is correct satisfactorily by tendon transfer operations (Srinivasan, Mukherjee and Subramaniyan, 1968; Srinivasan, 1978b; Malaviya, 1981).

Socio-economic rehabilitation

Majority of leprosy patients do not require any more than adequate treatment of leprosy, proper and effective counselling and prompt attention to complications.

There is, however, a proportion of patients who need addition measures for their socio-economic rehabilitation. These measures include assistance in the form of seed money as capital, vocational training, sheltered employment, job placement etc. Our aim as physicians, however, is to see that the necessity for such assistance is reduced to the inescapable minimum.

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