NEUROARThROPATHY OF THE FOOT IN LEPROSy

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Among 449 patients with leprosy, 40 had clinical and radiographic evidence of neuroarthropathy in 50 feet. These changes were classified into four types according to the joints first involved by major lesions: ankle (25 feet), midtarsal (15 feet), tarsometatarsal (7 feet) and subtalar (3 feet). The progression of joint destruction was different in each type, but despite the severe destructive changes seen in radiographs, the patients had relatively few complaints.

The muscles innervated by the peroneal nerve were severely paralysed in ankle and midtarsal types and it seems that, over a long term, repeated trauma and/or abnormal stress may lead to these types of neuroarthropathy. Neuropathy was less severe in the tarsometatarsal type of joint degeneration; the pathogenesis in this type seemed to be mainly direct trauma to the forefoot.

Leprosy, with infection of skin and nerves by Mycobacterium leprae, is one of the major causes of neuroarthropathy (Eichenholtz 1966). Leprotic patients develop peripheral neuropathy with a variety of secondary changes in the feet (Paterson 1961). A number of reports of bone and joint changes in the foot have included those which were the result of direct bacterial infection (Harris and Brand 1966; Warren 1971, 1973; Kularni and Mehta 1983). We have excluded such cases and have studied the incidence, clinical features, development and pathogenesis of true neuroarthropathy secondary to leprosy involving the foot.

MATERIALS AND METHODS

From the 449 leprotic patients at Ohshima Seishouen, 282 were randomly selected for clinical examination and a lateral radiograph of the ankle and foot. The age of these patients ranged from 36 to 94 years (mean 62.5 years), 177 were men and 105 women. The duration of leprosy ranged from 16 to 71 years (mean 42.5 years).

Bone and joint changes were common in the metatarsophalangeal and interphalangeal joints but, since most were secondary to skin ulceration, these lesions were excluded from the study. Patients with abnormal radiographs had a review of their case history and more detailed radiographs of the ankle and foot were taken, with anteroposterior, lateral and stress views. The diagnosis of neuroarthropathy of the ankle or foot was then made from the clinical and radiographic findings.

In these cases, the degree of neuropathy was evaluated by examining for sensory loss and the strength of four muscle sectors (tibialis anterior and the peronei; tibialis posterior and triceps surae). We developed a nerve score to quantify the strength of the muscles innervated by the peroneal and tibial nerves, using the five point MRC scale for each sector. The peroneal nerve score was obtained by adding the scores for the tibialis anterior and the peroneal muscles, with five points maximum for each. The tibial nerve score is the addition of strengths for tibialis posterior and for the triceps surae, again with five points for each. Thus, the normal value for each nerve is 10 points.

The radiographs of each case were reviewed and classified into three stages of change: Stage 1, development; Stage 2, coalescence; and Stage 3, reconstruction (Eichenholtz 1966).

RESULTS

Of the 282 patients, 109 had bone or joint abnormalities on the routine lateral radiograph. Of these, 50 extremities in 40 patients were diagnosed as showing neuroarthropathy from clinical and full radiographic review. In eight of these cases, ankle arthrodesis had been performed, but the diagnosis of neuroarthropathy could be made from the earlier radiographs. The incidence of leprotic neuroarthropathy of the ankle, mid and hind foot in our series was 14%.
**Classification of neuroarthropathy.** Leprrotic neuroarthropathy may be manifested at various sites and with various clinical features. We recognised four types according to the level of main joint which had been first destroyed (Fig. 1). These types were:

1. Ankle (25 feet).
2. Midtarsal, at the level of a Chopart amputation (15 feet).
3. Tarsometatarsal, at the level of a Lisfranc amputation (7 feet).
4. Subtalar (3 feet).

In some cases, many bones and joints were involved in the same foot. For these, the first joint to be destroyed was decided by reviewing the serial radiographs. The clinical features in the four types are summarised in Table I. Neuroarthropathy of the ankle was subclassified into the three stages described by Eichenholtz (1966).

**Ankle.** Twenty patients presented with 25 neuroarthropathic ankles, eight of which had already been arthrodesed. In 21 feet there had been some trauma (fracture in two feet, repeated sprain in 19). In four cases neuroarthropathy had developed within a year or two of anterior transfer of the tibialis posterior tendon for the correction of drop foot.

![Fig. 1](image)

Diagram to show the location of leprotic neuroarthropathy in 50 feet. The bold numbers indicate the major primary joint involvement in the series; those in parentheses are the numbers in which that level eventually became involved.

The onset of neuroarthropathy in most patients was shown by swelling with or without pain. At the latest review, swelling was seen in nine ankles, but only one patient complained of mild pain. Three ankles in three patients showed anterior instability and nine ankles in seven patients showed both anterior and varus instability.

![Fig. 2](image)

Radiographic changes during the course of neuroarthropathy of the ankle. The stages are those of Eichenholtz (1966), see text.
All patients had numbness below the knee joint. The peroneal nerve score was normal for three ankles and zero in those with complete drop foot. The average peroneal and tibial nerve scores were 2.3 and 8.1 respectively.

The radiographs of each case were reviewed and characterised as showing one of three stages:

Stage 1. The characteristic at this stage, seen in all cases, was the presence of a loose body. Dissociation of the tibiofibular joint was seen in 42%. Adjacent joints were not affected at this stage.

Stage 2. Dissociation of the tibiofibular joint (seen in 84%) and disruption of the anterior articular surface of the tibia (seen in 78%) were the typical changes at this stage. Neuroarthropathy had extended to the subtalar joints in 56% and to the midtarsal joints in 22%.

Stage 3. The characteristic was fusion of the tibiofibular joint (seen in 79%). The subtalar joint was affected more frequently than in Stage 2.

The progress of disease varied with each case and did not necessarily follow the sequence of these stages, as judged from the radiographs (Fig. 2). The duration of Stage 1 averaged about two years. Stage 2, usually longer than Stage 1, was very variable, but averaged four years. The radiographic changes showed definite progression in Stages 1 and 2, but there seemed to be little progress once Stage 3 changes were seen.

**CASE REPORT**

A 60-year-old man had repeated inversion sprains of the right ankle, with chronic swelling (Fig. 3a). One year later the radiograph showed dissociation of the tibiofibular joint and a loose body (Fig. 3b). Two and a half years later there was flattening of the talus and destruction of the tibial articular surface (Fig. 3c). Six years after onset of the symptoms, he had developed anterior and varus instability of the ankle joint with fusion of the distal tibiofibular joint (Fig. 3d).

**Midtarsal joints (Chopart).** Fifteen feet in 12 patients showed major changes around the midtarsal joints. Nine patients gave a history of trauma: two had had fracture of the neck of the talus and seven had had recurrent sprains. One patient had noticed spontaneous swelling of the foot after the anterior transfer of the tibialis posterior

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**Table 1.** Details of 40 leprotic patients with neuroarthropathy of the foot

<table>
<thead>
<tr>
<th>Main joint involved</th>
<th>Ankle</th>
<th>Midtarsal</th>
<th>Tarsometatarsal</th>
<th>Subtalar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>20</td>
<td>12</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>Number of feet</td>
<td>25</td>
<td>15</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>Age in years</td>
<td>63.7 (40-82)</td>
<td>59.2 (48-70)</td>
<td>58.3 (48-71)</td>
<td>66.3 (56-76)</td>
</tr>
<tr>
<td>Duration of leprosy in years</td>
<td>46.2 (34-60)</td>
<td>40.8 (27-52)</td>
<td>36.3 (23-46)</td>
<td>48.7 (41-59)</td>
</tr>
<tr>
<td>Duration of neuroarthropathy in years</td>
<td>12.8 (2-40)</td>
<td>22.9 (2-38)</td>
<td>16.9 (3-30)</td>
<td>17.0 (5-38)</td>
</tr>
<tr>
<td>Peroneal nerve score</td>
<td>2.3</td>
<td>2.1</td>
<td>8.4</td>
<td>6.7</td>
</tr>
<tr>
<td>Tibial nerve score</td>
<td>8.1</td>
<td>8.1</td>
<td>10</td>
<td>6.7</td>
</tr>
</tbody>
</table>
tendon. At onset, 14 feet were swollen and seven were painful. At the latest review, no patient complained of swelling, pain or obvious instability at the affected level, but most showed planovalgus deformity of the foot. All patients had numbness below the knee joint. The average of peroneal and tibial nerve scores were 2.1 and 8.1 respectively.

Three subgroups of changes in and about the navicular bone could be recognised: in five feet the bone was flattened but there was little or no change in the talonavicular joint. In another five feet the navicular bone had collapsed and shifted medially while the talonavicular joint was moderately affected (Fig. 4). In the remaining five, other adjacent joints were affected, but not the tarsometatarsal joints. Deterioration in the neuroarthropathic changes could be detected over a period of from two to 19 years in 10 feet, but in five the destructive process had ceased to progress.

**Tarsometatarsal joints (Lisfranc).** Seven patients showed major changes at this level. Three patients had suffered direct blows and had had fractures of the cuneiform bones, while four patients had had recurrent sprains of the forefoot without evidence of fracture. At the onset, all seven patients had complained of swelling and four had pain, but at the latest review none complained of either swelling or instability. All patients had numbness below the ankle joint. The peroneal nerve score varied (mean 8.4), but tibial nerve scores were 10 (normal) in all patients.

Radiographic follow-up ranged from three to 30 years. At first there was fragmentation, marginal sclerosis and narrowing of the joints. Destructive changes ceased after fusion of the joints in four feet; two feet showed arrest of the destructive changes without fusion, and one continued to deteriorate, with changes in adjacent joints (Fig. 5).

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**Fig. 4a**
Radiographs of a 50-year-old-woman with neuroarthropathy of the midtarsal joint. Figure 4a – When swelling appeared, the radiograph showed collapse of the navicular. Figure 4b – Ten months later.

**Fig. 4c**
Figure 4c – Three years later the medial side of the foot had collapsed further. Figure 4d – Eight years after Figure 4c there has been little radiographic change.
Subtalar joints. Changes in the subtalar joints followed incongruence caused by calcaneal fracture. This occurred primarily in three feet. The subtalar joints, however, are likely to be involved secondarily following other types of neuroarthropathy (Fig. 1). Symptoms in this type were very mild.

DISCUSSION
Charcot first described the destructive joint changes associated with tabes dorsalis in 1868 (Eichenholtz 1966; Resnik 1981). Since then many other causes for a "Charcot joint" have been reported. Progressive degenerative joint change due to central and peripheral nerve lesions are now termed "neuroarthropathy" (Resnik 1981).

Harris and Brand (1966) reviewed the bone and joint changes in leprotic feet and used the term "disintegration of the tarsus" because a bone injury or infection often dominated the picture. Destruction was rapid and progressive while infection was active but, once it subsided, these changes stopped. By contrast, truly neuroarthropathic changes tended to be slowly progressive. Since the pathology of these two types of change is obviously different, neuroarthropathic changes should be distinguished from infectious ones.

Our patients had had neuropathy for a long time with little evidence of active leprosy (bacillus positive rate 8.3%). Leprosy had been diagnosed an average of 33.4 years before the onset of neuroarthropathy in the ankle type, 17.9 years in the midtarsal type and 19.4 years in the tarsometatarsal type. Neuroarthropathy seems to result from long-standing neuropathy. The ankle type of neuroarthropathy was likely to follow peroneal motor loss with drop foot. With such a palsy, leprotic patients lacking protective sensation and muscular reaction are liable to suffer the repeated inversion injuries of the foot which possibly cause neuroarthropathy.

Most patients with the midtarsal type of lesion had a peroneal nerve score of zero. Total paralysis of the tibialis anterior concentrates stress on the talonavicular area during the push-off phase of walking, and this is one of the causes of navicular collapse (Kularni and Mehta 1983). Both the repeated trauma and the abnormal stress may contribute to the neuroarthropathy.

In the tarsometatarsal type, the neuropathy tended to be less severe; most patients had experienced a direct injury to the forefoot. This type of neuroarthropathy may be mainly due to direct trauma rather than to repeated trauma or abnormal stress.

The authors wish to express their appreciation to Drs Okada, Imaizumi and Hashizume for their advice in this follow-up study and encouragement in the preparation of this manuscript, and to Professor Ono for his helpful criticism and comments.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

REFERENCES