Lepr. Rev. (1972) **43**, 137-147

Management of Tarsal Bone Disintegration*

GRACE WARREN

Hay Ling Chau Leprosarium, Hong Kong

Tarsal bone lesions may occur in as high a proportion as 25% of leprosy patients. Some of these lesions develop into a progressive disintegration that results in marked deformity and increasing disability. For years this was considered to be part of the specific leprosy process and to be unresponsive to routine treatment methods. Recent investigations show that these lesions will heal in response to simple treatment methods, but require a considerable length of time. This paper describes these lesions, their diagnosis and treatment, with special reference to the clinical signs and symptoms for those who do not have facilities for radiographic examination.

Many leprosy patients, when the disease is first diagnosed, already have marked deformity and disability of the feet. The leprosy worker is confronted with the problem of making the best of what is left in order to try and give the patient feet that are ulcer-free and useful. However, when reviewing patients who have been under medical treatment for years, one realizes that although on admission the patient had "good feet" they are now deformed and require special care. Why does this progressive deformity occur, and what can we do to prevent it?

The common causes of progressive deformity are:

(1) Repeated minor ulcers and other trauma which slowly result in the loss of the tissue of the foot, especially the toes and the front of the foot. This trauma can usually be prevented in the co-operative patient by adequate education in the care of the feet and the use of suitable footwear.

(2) Gross sepsis and osteomyelitis; these may result from an accident, a burn, or neglected minor trauma, and cause marked loss of tissue, with scars that may contract and distort part of the foot. Education of the patient and instructions to report and treatment of all infections as soon as possible will minimize resultant deformity and loss of tissue. Treatment must include *adequate* rest, which is often neglected because of the accompanying diminution of pain perception.

(3) Progressive breakdown of bone (Paterson, 1961); this is not of necessity associated with sepsis but gradually allows increasing deformity to occur. For long it has been thought that these lesions are a part of the specific leprous process and that they are difficult or impossible to treat (Harris and Brand, 1966). However, recent investigations have shown that early diagnosis and active treatment will result in bone healing without deformity. The bones most commonly affected are the tarsal bones, though the adjacent metatarsal bases and the lower ends of the tibia and fibula may be secondarily involved. The condition is hereinafter referred to as tarsal bone disintegration.

The first two conditions are well known and articles on them are legion. The remainder of this paper is on the third cause, and is specifically written, bearing in

* Received for publication 31 July, 1972.

mind that many leprosy workers do not have radiographic facilities readily available.

Tarsal Bone Disintegration

In some leprosy patients progressive disintegration of bone appears to follow comparatively minor trauma. This usually occurs in persons who have a diminution of pain perception and so continue to retraumatize the bone during everyday usage. Having no warning of the increasing damage they do not rest the traumatized bone and thus encourage the body's attempts to repair the damage. This disintegration may be progressive until there is complete destruction of part of the foot. If the repeated trauma is prevented, healing of the damaged bones can occur (Warren, 1971b).

Clinical Findings

The earliest lesions present as a minimal swelling of the foot, with some heat over the affected area (Lennox, 1964); there is no obvious deformity of the foot and the patient does not usually complain of any pain or discomfort, but an abscess may be suspected (Fig. 1a, b) (Price, 1960).

Careful history-taking may reveal:

(1) An accident causing unusual trauma to the foot (tripping in a drain or over a stone is common) (Paterson and Job, 1964).

(2) A period of relative inactivity such as occurs during prolonged bed rest for intercurrent disease or chronic lepra reaction.

(3) A period of immobilization of a foot in plaster, such as is used for the treatment of ulceration of the foot in leprosy, followed by rapid resumption of walking. The immobilization may have been of the unaffected foot, nevertheless resulting in relative inactivity during the use of a plaster cast, but is more commonly of the affected one (Harris and Brand, 1966).

(4) A recent area of sepsis on the foot, though this may have already healed.

(5) The use of corticosteroids for leprosy, or intercurrent disease. In many countries corticosteroids can be purchased easily and are taken as analgesics, so that their use may not be immediately obvious.

(6) Recent lepra reaction, including neuritis, and commonly associated with paralysis or paresis of the affected foot, or with swelling of the foot.

However, in many instances none of these factors is obviously present and the patient denies any possible cause for the heat and swelling.

In more advanced cases some crepitus (Lennox, 1964) may be palpated over the point of maximum heat and swelling and a flattening of the arch of the foot may be observed when the two feet are placed sole to sole. This flattening of the arch is a very useful sign (Fig. 2a, b).

Further bony disintegration may result in abnormal mobility of the affected area—usually the talo-navicular area. If excessive mobility (Lennox, 1964) has been present for some time there may be no obvious heat, though residual swelling and deformity may be present. The patient could still walk on the ankle illustrated in Fig. 3, but he complained of some instability! In these later cases there is usually little doubt regarding the diagnosis, and full treatment should be instituted without delay if a functional foot is to be achieved after healing.

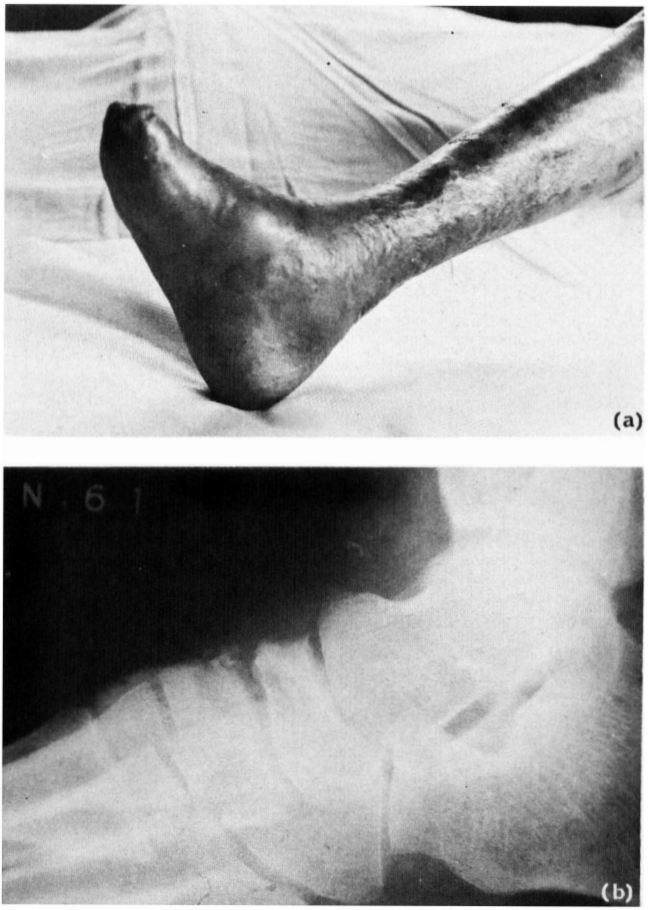

Fig. 1(a). Swelling of the foot that may occur with an early naviculo-cuneiform lesion; (b) radiograph of (a). The lesion commenced as a fracture of the talus and calcaneum 5 years illustrated in Fig. 1(a).

The progress of disintegration varies greatly in different patients. In one patient with a known fracture sustained 6 months earlier there was still no appreciable deformity and minimal bone disintegration. In another, there was complete loss of the talus, with disorganization of the ankle within 6 months of his initial complaint of "slipping into a gutter".

If radiographic examination is possible, it is the ideal method of diagnosing and controlling the treatment of this condition, but in its absence adequate treatment that will prevent permanent disability is still possible.

For radiographic diagnosis two views are really necessary.

First, AP or preferably APO (which more clearly defines the anterior tarsal bones). The film should be placed and the exposure adjusted so that a clear view of the talus head is obtained. This means the phalanges of the toes will be over-exposed and not well visualized.

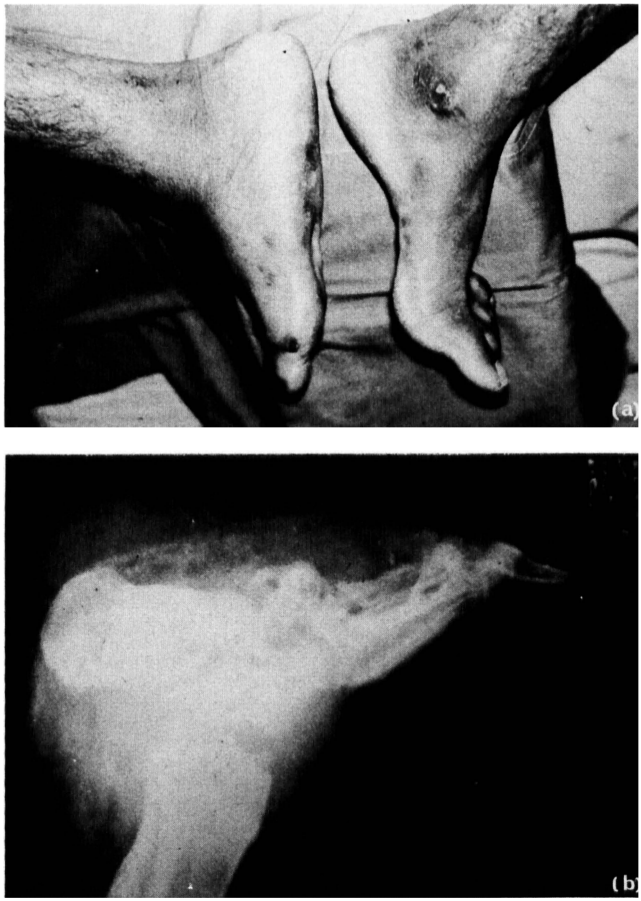

Fig. 2(a). Flattening of the arch of the affected foot; (b) marked radiographic changes of affected foot shown in (a).

Second, a true lateral view of the calcaneum, with the film placed so that all the tarsals and the lower portion of the tibia are visualized. A *standing* lateral view gives the best position for future comparisons. If deformity of the ankle is present care must be taken to get the true lateral of the calcaneum and not of the ankle joint. These two views should reveal all lesions, though in a grossly distorted ankle some adjustment of this position may be needed to define the affected bone and an AP view of the ankle may also be advised.

Early lesions of the talus and navicular are most easily diagnosed from the lateral view, but those of the middle and lateral cuneiforms and cuboid are seen earlier on the APO view. With careful placement it is possible to get the APO and lateral views of one foot on to one 8 x 10 in X-ray film (Fig. 4). If films are readily available, it is desirable to radiograph both feet so that the abnormality can be compared with the normal foot. Ideally, every patient should have his feet radiographed at the time of the diagnosis of leprosy in order that any early bone

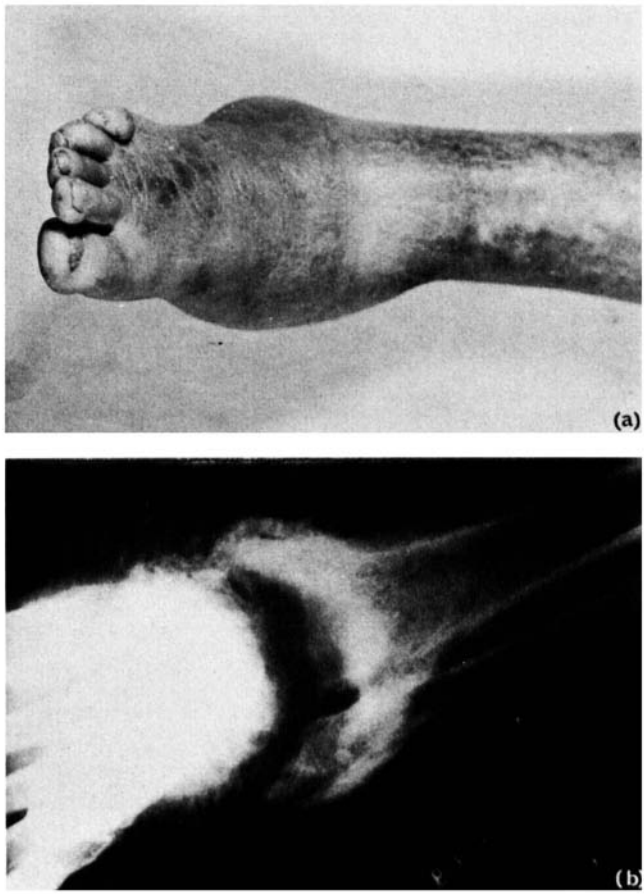

Fig. 3(a). Soft fluctuant swelling of the ankle area without heat or obvious inflammation; (b) radiograph of (a). The lesion commenced as a fracture of the talus and calcaneum 5 years earlier.

lesions can be detected and treated before real deformity occurs and to provide a base line for later comparisons.

The early and less disabling lesions may appear as:

(1) a vagueness of outline of one or more bones, with or without a definite change in shape; (2) a frank fracture (Paterson, 1961); sometimes attention is drawn to it by surrounding osteoporosis; (3) a portion of the medullary bone appearing to be squeezed out through the fracture line (Fig. 5); (4) a change in shape of a bone without any obvious break in the continuity of the cortex (especially the talus) (Fig. 6a, b).

Late lesions may appear as a gross change in the shape of one or more bones, with the loss of adjacent joint spaces. The residual bone may be fragmented and the stress of walking may have displaced the fragments, so that the remaining bones are impacted and the normal architecture of the foot is lost. Every degree

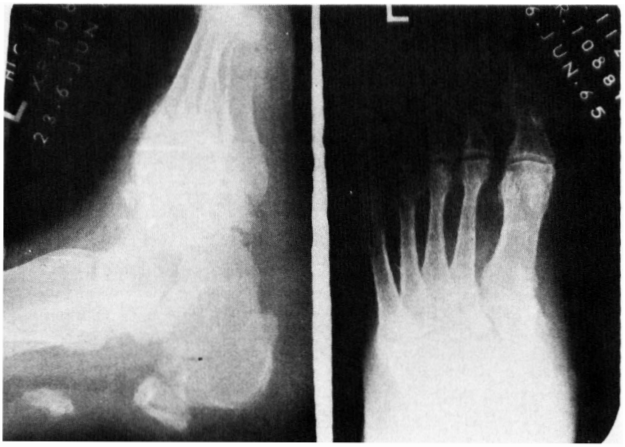

Fig. 4. Placement of APC and lateral views on one 8 x 10 in film. A deformity of calcaneum and another of the talo-navicular area (with pin *in situ*) are visualized.

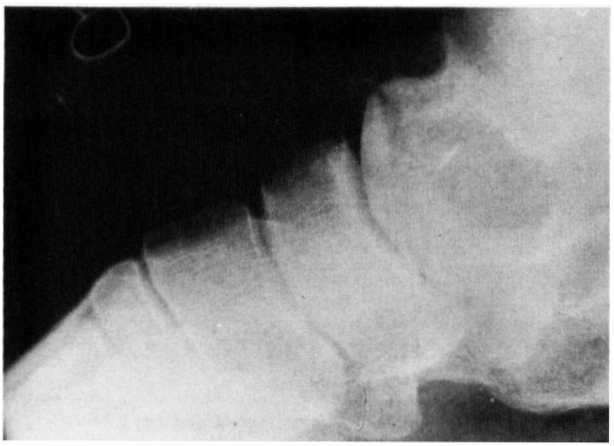

Fig. 5. Portion of medullary bone being squeezed out through a crack in the cortex of the talus. This lesion progressed to complete loss of the neck of talus and collapse of the head when unrestricted walking was allowed for 6 months after a short period of plaster immobilization. Healing eventually required 18 months' immobilization.

of deformity may be present. The still active bone lesions will have a hazy appearance. Healed lesions will usually appear sclerotic, with smoothed-off edges (Fig. 7a, b, before and after PoP). At surgery it has been found that this bone is very dense and hard. In practice this healed sclerosed bone has not been seen to fracture or to disintegrate.

Management

The treatment for all stages of tarsal bone disintegration is immobilization in the functional position for an adequate period of time to allow complete bone

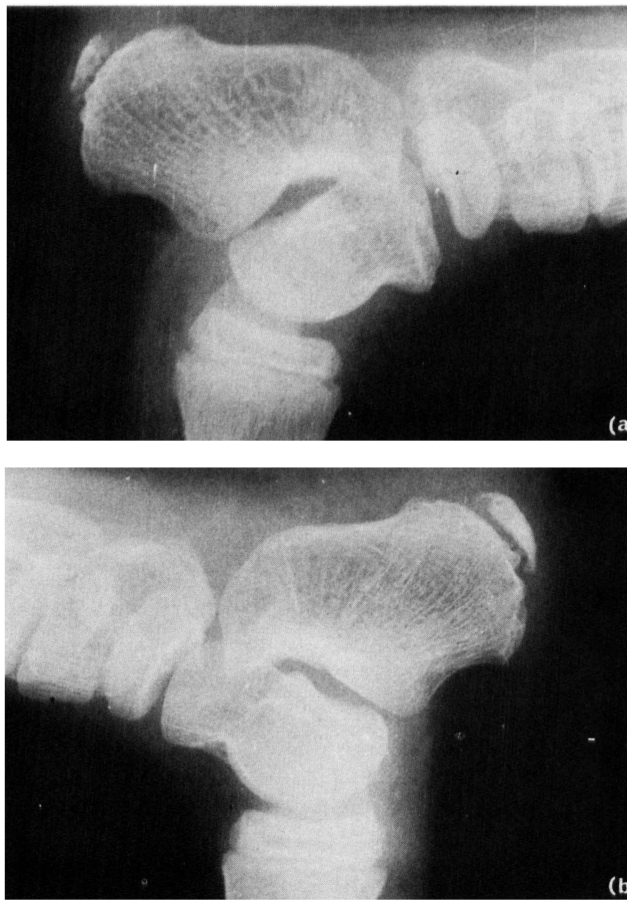

Fig. 6(a). Deformed talus navicular bone in foot of 8- to 10-year-old Chinese boy; (b) normal foot for comparison.

healing to occur. This is usually achieved by use of a below-knee plaster-of-Paris (PoP) walking cast. Ideally the length of time should be determined radiographically, but in the absence of this the following method will prove effective.

The earliest lesions are difficult to differentiate from fractures and sprains. Even radiographic evidence of a stress fracture may not be apparent for 4 to 6 weeks. If a patient presents with a slightly warm, swollen, undeformed foot and no definite history, it is advisable to support the foot with a crêpe bandage and give diuretics for a few weeks, while allowing the patient to continue reasonable activity. If the symptoms do not subside within 4 weeks, then it should be assumed that a bone lesion is present, even if radiographic examination is not possible. If radiographic examination is possible it should be done every 3 to 4 weeks until all symptoms have subsided, or a bone lesion is apparent.

Once it is decided to treat the lesion as a tarsal bone disintegration a well-fitted (PoP) walking cast should be applied, taking special care to mould the plaster to

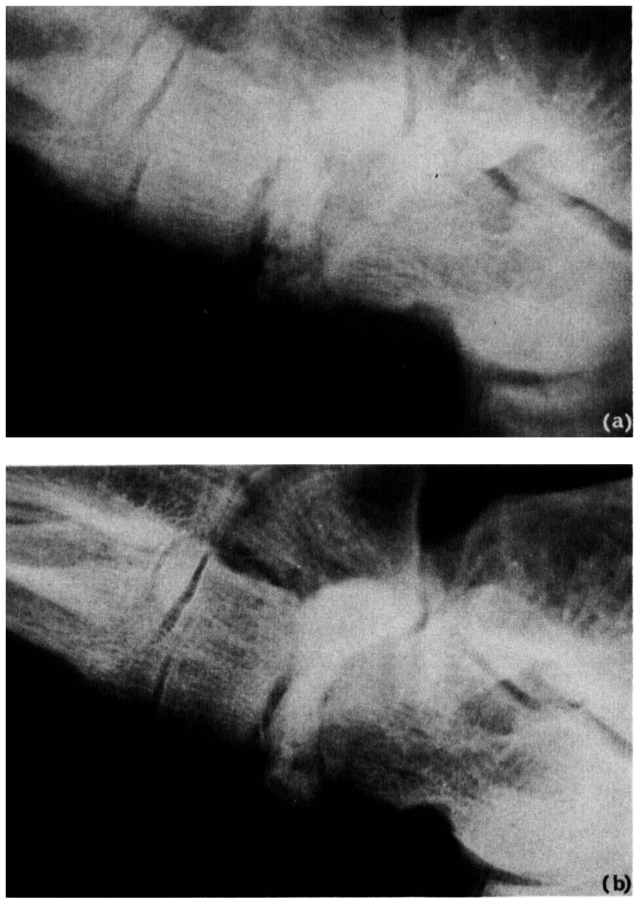

Fig. 7(a). Breakdown in navicular bone; (b) healing of navicular bone after 6 months' immobilization.

the shape of the foot and to hold the foot in a functional position (ankle at 90° without inversion of the heel) (Warren, 1971b).

If the bone lesions involve the body of talus and the calcaneum immediately below the talus, the weight should be carried through a Bohlers iron from the tibia. The leg portion of the plaster needs to be extra well fitted to do this (Warren, 1971). For bone lesions at other sites a standard wood or rubber rocker can be used, or a flat soled plaster can be applied to be worn in conjunction with a flat sandal. In the foot with a doubtful lesion, this plaster can be kept on for 2 to 3 months; at the end of this period trial walking should show if it was a true bone lesion or not. For a definite lesion, the plaster can be changed at 3 months to allow radiographic studies when these are available; the plaster can then be reapplied for another 2 to 3 months before further radiographic studies, and trial walking carried out if the radiographs are satisfactory.

The early lesion requires 4 to 6 months for full healing, so if radiography is not

available it is advisable to maintain plaster immobilization for about 5 months before allowing trial walking. Changing of the plaster should be well supervised. If radiographic studies are possible it is important to ensure that the patient does *not* walk while waiting at the X-ray department, as he may damage the healing bone and delay further healing. Also, if at all possible, the plasters should be applied by the medical officer in charge himself so that the optimum position may be achieved. This is especially important when there is mobility of the foot, as in the more advanced cases, and a poorly moulded plaster may increase the deformity.

Trial walking is a graded method of return to unsupported walking.

As it is impossible to assess, on radiological evidence alone, if healing after disintegration is complete, the following routine has been formulated. It does reveal most cases of incomplete healing and at the same time provides a gradual return to activity that assists in recalcification of the osteoporotic bones and helps reduce the possibilities of stress fractures and further episodes of disintegration. Trial walking is also used at Hay Ling Chau, with modifications, on all occasions when normal ambulation is being resumed after prolonged non-use of a foot, such as after ulceration, reaction, surgery, or complete bed rest.

Method

(a) The affected foot is firmly bandaged; (b) suitable shoes with resilient insoles are laced on to the foot; (c) walking around the bed is then allowed for 3 min on three occasions on the first day; (d) after walking the foot is checked for heat and swelling. This procedure is repeated daily, provided no heat or swelling develops, but the time is increased gradually, to 5 min on the second day and 10 min on the third day, so that at the end of a week the patient is walking about, in the hospital, for 30 min three times a day.

If swelling of the foot occurs, or there are other indications of impending trouble, the increases are taken more slowly and diuretics may be given, but if the swelling subsides before the next day it is usually only "travel oedema" and the use of the crêpe bandage support helps to prevent this. If heat over the damaged bones occurs and does not subside overnight the bones should be regarded as not fully healed and complete immobilization resumed for a further 6 to 12 weeks.

Trial walking also helps to prevent breakdown of the skin of the sole such as frequently occurs when a patient with an anaesthetic foot resumes walking after a prolonged period of complete immobilization or bed rest. If the patient has adequate muscle action, he can start using weights in conjunction with trial walking to stimulate movement and circulation without the risk of a fracture. This commences with half-pound (230-g) resistance to dorsiflexion and the weight gradually increased as strength returns.

Advanced Lesions

In more advanced disintegrations the diagnosis is more obvious. If heat and/or swelling are still present, healing can be expected. If mobility has already occurred it is wisest to allow 9 months of immobilization before trial walking when radiographic control is not available. The position of the foot during each plaster application is most important. If the foot cannot be moulded into a functional position it may be advisable to consider surgical reconstruction, using internal

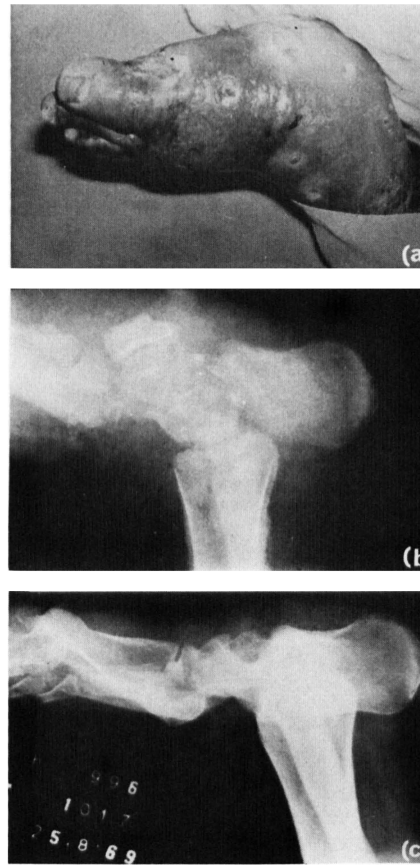

Fig. 8(a). External view on admission of foot shows multiple discharging sinuses; (b) radiograph on admission shows osteomyelitis and disintegration; (c) radiograph some 12 months later shows bone healing well advanced.

fixation. Healing of these bones does occur after surgery if complete immobilization is achieved. A period of 9 to 12 months is required for healing of a mid-tarsal wedge osteotomy, but a talo-calcaneo wedge will usually heal in 4 to 5 months.

If there is no sign of heat or swelling and the deformity and/or mobility has been present for a long time without change (over 6 months), it can be assumed that any lesions that were present have healed. On radiographic examination they will be seen to be smoothed off and sclerotic. Simple immobilization will do little, if anything, to improve stability and reduce disability unless surgical intervention precedes the immobilization. It may be necessary to use a bone graft from the iliac crest to make up for a deficit in bone bulk. The hard sclerosed bone must be chiselled out until a bleeding surface is displayed and complete immobilization maintained for up to 12 months, or possibly even longer. Any infection may lead to rejection of the graft and will delay fusion, but provided the foot is held completely immobilized at all times, in the position of function, a favourable final

outcome can be hoped for. This sometimes poses problems and requires some ingenuity to immobilize and yet at the same time allow for dressings of discharging sinuses.

In the foot in which bone disintegration is complicated by sepsis, the same principles hold. Immobilize in the optimum position while treating the infection with local packs and antibiotics, and a useful foot can often be salvaged (Fig. 8a, b and c). But REST is essential—and because of the diminution of pain perception the patient will not rest unless forced to. Even one weight-bearing step taken at a plaster change may fracture the newly formed callous and delay healing by many months.

Comments

Disintegration of the bones of the foot of a leprosy sufferer may progress until the foot is a useless member and a real disability. However, no matter how severe the lesion is when first diagnosed, it is usually possible to achieve healing of the remaining bones. Provided that destruction has not progressed too far, the remainder of the foot can become a useful member again, though this may require much time and ingenuity on the part of the medical workers.

Tarsal bone disintegration is no longer a condemnation to deformity or amputation, especially if detected and treated early.

Minor foot trauma is often unnoticed because of the diminution of pain perception, but we need to teach our medical personnel, and the patients themselves, to watch for early signs and symptoms so that early lesions of all types can be effectively treated and more severe deformity and disability prevented.

References

Harris, J. R. and Brand, Paul W. (1966). Patterns of disintegration of the tarsus in the anaesthetic foot. *J. Bone Jt Surg.* **48B**, 4-16.

Lennox, W. M. (1964). A classification of leprosy foot deformities. *Lepr. Rev.* **35**, 245-249.

Paterson, D. E. (1961). Bone changes in leprosy; their incidence, progress, prevention and arrest. *Int. J. Lepr.* **29**, 393-422.

Paterson, D. E. and Job, C. K. (1964). Bone changes and absorption in leprosy. *Leprosy in Theory and Practice*, 2nd ed., Chapter 26. (Eds R. G. Cochrane and T. F. Davey.) Bristol: J. Wright and Sons Ltd.

Price, E. W. (1960). Studies in plantar ulcers in leprosy. *Lepr. Rev.* **31**, 97-103.

Warren, Grace (1971a). Plaster casts. *Lepr. Rev.* **42**, 55-59.

Warren, Grace (1971b). Tarsal bone disintegration in leprosy. *J. Bone Jt Surg.* **53B**, 688-695.