Lep. Rev. (1964), **35**, 4A, 259/266

The Etiology and Natural History of Plantar Ulcer

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ETIOLOGY

The lesion called 'plantar', 'perforating' or 'trophic' ulcer is not peculiar to leprosy and is known to occur in several diseases in which there is a neuropathic lesion of the foot, but in which walking is still possible.

It does not occur when walking is not possible; and for these reasons it is seen in tabes, syringomyelia, and diabetic neuritis, but is uncommon in such disabling conditions as spina bifida and transverse myelitis where the patient may be bedridden. In leprosy, the disease is the cause of the nerve deficit which produces the plantar ulceration; leprosy does not cause ulcer directly.

The neuropathic foot is deficient in varying degree of motor, sensory and autonomic function; and all of these play a part in weakening the foot to the extent that walking causes ulceration. The separate deficiencies have been studied by several workers, and the relative importance of each has been discussed elsewhere (PRICE, 1959, 1961). The treatment that gives consistently good results is based on the assumption that the predominating factor in causation is the tissue damage due to the mechanical strains of walking.

The way in which this damage occurs is now well known and is described by several recent writers (ANDERSEN, 1961; ROSS, 1962; ZAMIDIO, 1963) and in the new Edition of COCHRANE and DAVEY, *Leprosy in Theory and Practice* (Wright: Bristol, 1964).

In summary, the crushing effect of body weight on the walking foot is added to a slipping that occurs between the bony framework of the foot and the ground beneath as each step is taken. This slipping does not occur in a foot with competent musculature; but when sensory and motor loss is present, the loss of control by the intrinsic musculature allows the soft tissues to be ground between bone and walking surface beneath, so that tissue necrosis takes place. The necrotic material slowly tracks to the surface, breaks through the skin and an ulcer is formed. The distribution of these ulcers is described in a later paper and is recognized to be that of the points of maximum walking pressure.

THE NATURAL HISTORY OF PLANTAR ULCER

The course of the lesion from its earliest beginning to frank ulceration can be readily observed whenever patients are submitted to regular weekly inspection. The duration and the ultimate permanent disability depend on two factors – the extent of pyogenic infection of the deep tissues, and the degree of collapse of the architecture of the foot which takes place.

In limited cases, the lesion may consist of a chronic ulcer with bouts of acute pyogenic infection; in more advanced cases, the attachment of the ligaments of the foot may be involved and collapse takes place. When collapse of the foot occurs, the primary ulcer may heal spontaneously and it is then often followed by a new ulcer at the prominent area of the sole. This latter ulcer may then persist indefinitely and even produce total destruction of the foot.

The natural history may therefore be considered in four phases:

1. Primary tissue damage
2. Primary plantar ulcer
3. Collapse of foot
4. Appearance of secondary ulceration.

1. *Primary Tissue Damage*

The sequence of events leading to ulceration passes through two stages that have been described as the *pre-ulcerative* state and the *necrosis blister.*

The *pre-ulcerative* state consists of localized swelling, deep tenderness, and evidence of deep oedema, such as spreading of adjacent toes. The disability is not severe enough to make a patient seek treatment, so that it will not be observed apart from regular foot-inspection. The sites affected are the pads over the metatarsal heads, the base of the fifth metatarsal, the heel, the interphalangeal joint of the big toe (and sometimes of the second toe) and the pulps of the toes. The time from the earliest symptom until the appearance of the next stage (the necrosis blister) is four to six days. This is the most favourable time for treatment and the lesion will resolve in a few days either by bed-rest or by the use of footwear with rigid sole and soft insole. Untreated, it passes inevitably into the next stage.

The *necrosis blister* is observed when the necrotic material from the deep tissue approaches the skin-surface. It is seen characteristically as a fluid collection, sometimes bloodstained, at the border of the hard skin of the sole. This means that it is seen either at the lateral or medial borders of the foot, or in the fold between the toes and the metatarsal pads. The sites are indicated by the edge of the shape left by a footstep in sand. The blister is largest at the heel, where it may contain 6–8 cc of sterile necrotic fluid. It is smallest at the anterior border of the metatarsal pads, under the toes where it may be mistaken for a banal or fungal infection. When the necrosis blister tracks to the surface directly through the hard skin of the sole, as it sometimes does, the swelling is hardly observed until the necrotic material is subcuticular; in this case the resulting lesion is collar-stud in shape and will give rise to a collar-stud type of ulcer. The necrosis blister rapidly becomes an ulcer if untreated; under treatment, it resolves in 10 days.

Both the pre-ulcerative state and the necrosis-blister are illustrated in the new edition of Cochrane and Davey.

2. *Primary Plantar Ulcer*

The sites of primary plantar ulcer are those of the previous necrosis blister, but it is not uncommon for ulcers over adjacent metatarsal heads to fuse and form a long ulcer across the metatarsal pads. The multiple ulcer on adjacent toe-tips is really one ulcer spread over several toes.

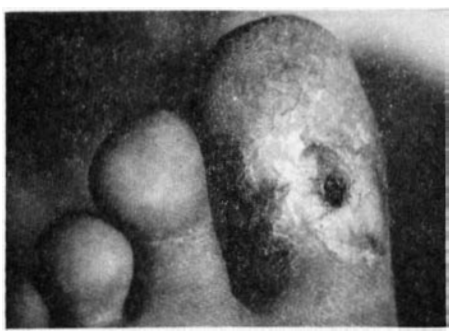
The importance of the ulcer, insofar as it concerns the future of the foot, can be considered as follows:

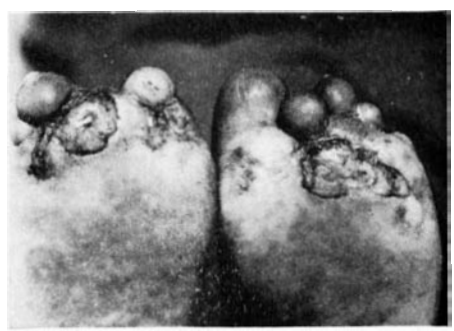
The most dangerous ulcer is that over the base of the fifth metatarsal.

The next is that of the heel, then that over the first metatarsal head.

The least dangerous to the foot are the toe-tip ulcers.

The reasons for this will be seen in the section on collapse of the foot.





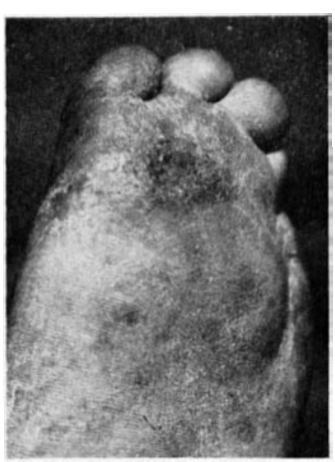


FIG. 1*a* (*top left*)

The Healing Process during Treatment with Footwear with Rigid Soles

(a) Stage 1. Diminution of exudate.

This ulcer, under the interphalangeal joint, was 2 cm. in diameter with considerable exudate, 10 days previously. It is now dry and epithelium is growing in rapidly.

FIG. 1*b* (*top right*)

(b) Stage of Epithelial Cover.

These four ulcers epithelialized under a petroleum jelly dressing within two weeks, using footwear with rigid soles. Epithelial cover proceeds rapidly if footwear is worn consistently.

FIG. 1*c* (*left*)

(c) Stage of Consolidation.

Tissue repair progresses under the epithelial cover, until the superficial scar resembles the surrounding skin. This ulcer healed within four weeks, after being open for nearly two years. Note the deformity caused by a previous metatarsectomy which made it difficult to fit footwear.

The stages by which the ulcer progresses have been described in several papers, so that the present occasion is taken to demonstrate the pathology

in reverse, so to speak. This can be observed by watching the healing that occurs when an ulcer is treated without any local application, except that the patient wears footwear with rigid sole. The following stages are recognized:

(i) Cessation of exudate (Fig. 1a).

The first sign of healing is a lessening of the exudate and this will be obvious within 24 hours, and is independent of pyogenic infection. It represents the beginning of deep-tissue repair.

(ii) Stage of epithelial cover (Fig. 1b).

As soon as exudate begins to lessen, epithelium rapidly grows in from the edges of the ulcer. Epithelial cover is replaced with surprising rapidity and defects of 2–3 cms can be completely covered within a week.

(iii) Stage of Consolidation (Fig. 1c).

When epithelialization is complete, the risk of secondary pyogenic infection ceases, but the 'ulcer' will rapidly recur if footwear is abandoned. Treatment – which in effect means continuation of the use of rigid-soled footwear – must be continued so that the scar consolidates in the deep tissues; at the same time, the superficial tissues and skin begin to take on the appearance of normal sole. This may be so effective that the site of ulceration may be difficult to recognize.

It is obvious from the above that a satisfactory method of treatment for uncomplicated plantar ulcer is the use of footwear with rigid sole. This is, in fact, what the writer uses in all such cases, the ulcer being covered with simple vaseline dressing to prevent soiling of the footwear.

3. *Collapse of the Foot*

The shape of the foot, which is commonly referred to as its arches, is maintained for the most part by the shape of the bones and the very strong

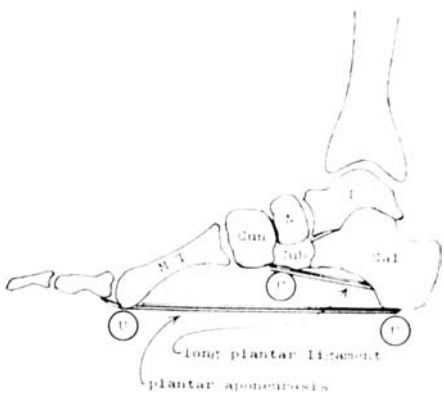


FIG. 2

Plantar Ulcers and the Ligaments of the Foot

Note that the common sites of ulceration overlie the points of attachment of the important plantar ligaments of the foot. The *heel ulcer* may involve the attachment of both the plantar aponeurosis and the long plantar ligament. The *mid-lateral ulcer* overlies the anterior attachment of the long plantar ligament; it is also in close proximity to the series of tarso-metatarsal joints and to those of the tarsal joints leading from the cubo-calcaneal region. This ulcer is therefore the most potentially dangerous of all plantar ulcers. The *ulcers over the metatarsal heads* do not seriously jeopardise the anterior attachment of the plantar aponeurosis because of the multiple attachments of the latter.

ligaments especially the plantar aponeurosis and the plantar ligaments, long and short (Fig. 2).

Muscular force, applied through tendons, is used mainly to prevent undue strain on these ligaments, but even complete muscular palsy does not cause collapse of the foot. The important ligaments are attached as follows: the plantar aponeurosis stretches from the calcaneum forward to the heads of the metatarsal and bases of the phalanges, and it will be observed that there is a multiple attachment in front.

The important long plantar ligament passes laterally from the calcaneum to the under-surface of the cuboid and adjacent bones. It will be seen at once that plantar ulcers lie over the attachment of these ligaments, and it will be recalled that the breakthrough of a necrosis blister from the deep tissues to the skin will expose these ligamentous attachments almost immediately to the risk of pyogenic infection.

The further steps in the natural history of a plantar ulcer that is not healing then proceeds as follows: osteoporosis with direct infection of bone where plantar ligaments are attached – partial detachment of underlying ligament – collapse of arch – projection of abnormal bony prominence in sole of foot – friction-pressure ulceration over new bony prominence.

It should be observed that collapse may follow the osteoporosis that follows longstanding disuse atrophy of a foot that has borne an ulcer for years; this is so whether or not there is direct pyrogenic infection of the bony attachment of the ligament.

The secondary ulcers that may occur depend on the type of collapse that has taken place and it is helpful to describe lateral collapse, medial collapse and total collapse of the arches of the foot. These may occur without resulting in secondary ulceration, but as the occurrence of this special type of ulceration emphasises the pathology, the types of collapse are described in the next section.

4. *Secondary Ulceration*

As the foot is progressively weakened by long infection or osteoporosis, the arches are progressively flattened by the weight of the body. Clinically, this event is noted by the projection, in the sole, of bony prominences that do not normally occur. The skin overlying these prominences may remain intact, but often superficial ulceration, due to friction-pressure, follows and then remains as a chronic or permanent lesion on the presenting surface of the sole of the foot. This ulcer has been called 'static ulcer', in distinction from the 'dynamic ulcer' which occurs at the beginning of the present clinical history of plantar ulcer.

The *lateral arch* depends largely on the integrity of the long plantar ligament and when it collapses it allows the following bony prominences to appear in the sole – the tuberosity of the cuboid and the anterior tubercle (keel) of the calcaneum. These two fall roughly in a line from the lateral tuberosity at the posterior part of the calcaneum at the heel, to

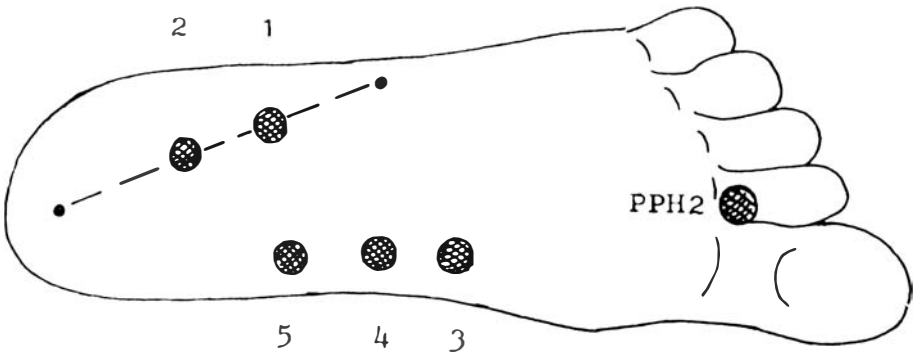


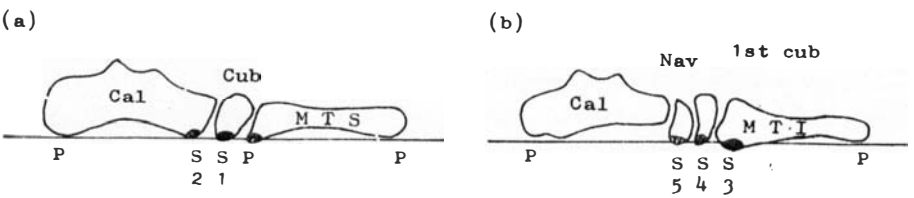
FIG. 3

Plantar Ulceration in Collapse of the Longitudinal Arches of the Foot

The diagram lists the sites of plantar ulceration which represent collapse of the longitudinal arches as follows:

1. Overlying the tuberosity of the cuboid.
2. Overlying the anterior tubercle (keel) of the calcaneum.
3. Overlying the base of the first metatarsal.
4. Overlying the medial cuneiform.
5. Overlying the tuberosity of the navicular.

PPH2 (Proximal Phalangeal Head 2) represents the ulcer that sometimes occurs over this bony prominence when the second toe attemps to take over the walking function of the big toe. This occurs when there is failure of the intrinsic musculature of the big toe.



(a) Collapse of lateral arch.
P represents primary plantar ulceration.
S represents secondary ulceration following collapse of the arch.
(b) Collapse of medial arch.
The numerals refer to the bony points on the diagram of the sole of the foot.

the tuberosity on the base of the fifth metatarsal (Fig. 3). An ulcer due to these prominences will lie along this line (Fig. 4).

The *medial arch* is much longer than the lateral and is supported by many structures; but the plantar aponeurosis and the many ligaments under the tarsal joints are mainly responsible for its shape. Detachment of one or more slips of the anterior attachment of the plantar aponeurosis is not likely to allow collapse of the arch, because the others remain intact. In fact, ulceration over one or more metatarsal heads does not usually lead to collapse of the foot.

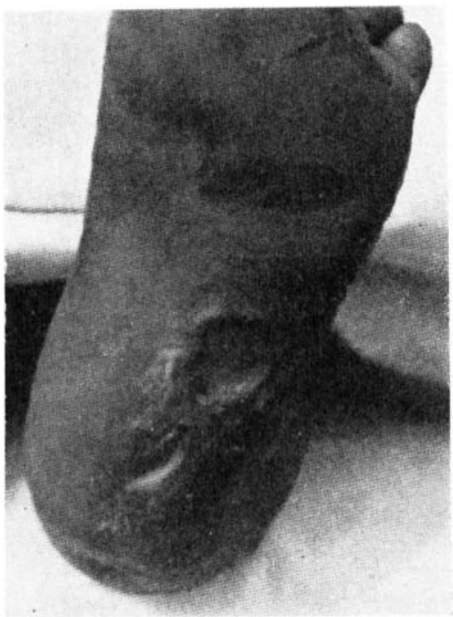


FIG. 4
Collapse of the lateral arch results in ulcers along a line from the base of the fifth metatarsal to the lateral half of the heel. In this case, the calcaneum is partly destroyed and the ulcers overlie the tuberosity of the cuboid and the keel (anterior tubercle) of the calcaneum.

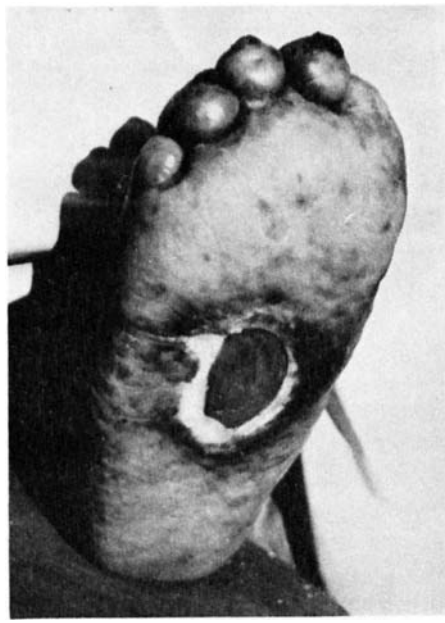


FIG. 5
Secondary Plantar Ulcer
This lesion, sometimes called friction-pressure ulcer or 'static' ulcer, to distinguish it from the 'dynamic' ulcer caused by walking on a mobile foot, occurs on the prominence of a foot that is totally collapsed.

The arches have all given way and the keel of the calcaneum underlies the ulceration. This is often called a 'boat-shaped' foot.

However, ulceration of the heel often leads to weakening and even detachment of a major part of the posterior attachment of the plantar aponeurosis. Collapse of the medial arch then allows new bony prominences to appear in the sole, and these again are in a roughly linear order proceeding medially forwards from the tuberosity of the calcaneum; these are the tuberosity of the navicular, the medial cuneiform and the base of the first metatarsal bone (Fig. 3). The overlying ulcer, if it occurs, demonstrates this clearly.

When both arches, lateral and medial, collapse, both linear ulcers may occur together. However, it is more usual for total collapse of the foot to follow a different course.

Total collapse of the foot allows the foot to take up, passively, the shape of a rocker which closely approximates to that which is carved on to the sole of wooden rockers. It is often called the 'boat-shaped foot'. The

prominence in the sole is the anterior end of the calcaneum and is mainly the keel of the bone.

An ulcer then appears to be directly central in the sole (Fig. 5). If ulceration does not occur, the collapse of the foot is total and if it is painless it can produce a surprisingly good rocker to the foot. It is often observed that as the rocker develops, any existing true ('dynamic') plantar ulcer will heal spontaneously. In effect, the body has achieved what we endeavour to do by using special footwear – it produces a state in which the strain of walking no longer falls on the front of the foot at its maximum pressure; in consequence, primary ulcers heal spontaneously.

DISCUSSION

The description of the natural history of plantar ulceration in leprosy shows that a small lesion of the foot may, if untreated, progress inexorably to total collapse of the foot. If this latter condition is also crowned by a chronic secondary ulceration, the foot may be totally lost as far as function and use is concerned. In addition, it is clear that the cause of the initial ulceration is a mechanical upset that can be controlled. In fact, medical knowledge of the lesion is such that every disabled foot in leprosy should indicate lack of sufficient care on the part of the patient or of the medical attendant. With the co-operation of both, leprosy should run its course to final arrest by therapy, without any major deformity or disability of the foot other than the loss of anesthesia (which may be inevitable) and some motor palsy (which can be compensated).

SUMMARY

1. The major factor in the etiology of plantar ulcer is mechanical.
2. The natural history of the lesion is described in detail from its earliest beginning to final destruction of the foot.
3. It is emphasized that plantar ulceration can be prevented, can be arrested at whatever stage it has reached, and should never be allowed to produce the deformed and defective feet that are still only too common in areas where the disease of leprosy is endemic.

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