PLANTAR ULCER IN LEPROSY: A REVIEW OF THE LITERATURE, 1890-1960

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Introduction

Recently there has been renewed general interest in plantar ulcers, and in their control as part of the process of rehabilitation of leprosy patients, and this paper will present a critical review of the literature to aid this work. We found *confused terminology* in the early papers on plantar ulcers. By some, any ulcer of the extremities in a subject of leprosy is called "leprotic". Others used the term "trophic" but included ulcers evidently hypostatic, and some even included the superficial ulceration of lepromatous lesions. Today the plantar perforating ulcer makes up most of the problem and it is found mainly in the tuberculoid and borderline kinds of leprosy. In our present review we have not included papers on the orthopaedic and plastic surgical procedures used in treatment of the complications of plantar ulcer. The modern literature on leprosy we found to begin with the recognised leprosy journals (Japanese journals should be included but were not available to us at the time of this study), namely *Leprosy in India* (1929), *Leprosy Review* (1930), *International Journal of Leprosy* (1933), *Revista Brasileira de Leprologia* (1934). Papers were few and scattered before 1930. The Tropical Diseases Bulletin, which began in 1912, gave a very useful service by its summaries. But as early as 1900 Lepra, of Biblioteca Internationalis, Leipzig, had also performed this function. In 1890 and 1891 appeared the Journal of the Leprosy Investigation Committee, which seems to be the earliest publication.

The Years 1890-1937. The Journal of the Leprosy Investigation Committee for 1890 has the earliest reference to specific treatment for plantar ulcer. It referred to the practice of stretching the enternal popliteal or great sciatic nerve. ARONGOM in 1905 stretched the posterior tibial nerve for the treatment of perforating ulcer. ROY (1934) was the first to recommend metatarsectomy, and GUIDA (1937) the first to report on therapy using a presumed vasodilator (acetyl choline). ARANGOM as above and SANDES (1913) described the "surgery of leprosy" but with little notice for plantar ulcers beyond the remark of SANDES that "nearly all lepers have ulcers".

The Years 1938-1960. Modern literature on plantar ulcers begins with a study by BECHELLI in 1938. There have been more than 90 papers since then, but many describe a few cases treated without controls and claiming good success. It is rare for the clinical condition and natural history of the lesion to be described. It is only in recent years that a serious attempt has been made to describe plantar ulcer.

Clinical Studies

In 1938 BECHELLI and colleagues described clearly from a study of 1600 cases in Brazil the development of the lesion. He noted that the distribution of plantar ulcers corresponded to "walking pressures" and concluded that the cause was "nervous", and secondarily mechanical stress. In the Compendio de Leprologia (1951) he repeated these conclusions. There seems little progress in the 20 years since this paper. COCHRANE (1947) gave a full account of the state of knowledge then, and clinically there was no further advance on BECHELLI. In recent years BRAND has referred to the subject, but there is no recent paper specifically studying the clinical picture until PRICE (1959) who gave an analysis of 2400 patients in Nigeria and developed the knowledge of the natural history of plantar ulcer, laying stress on the significance of the "necrosis blister". In 1960 LANGUILLON and colleagues followed by reporting on 3000 patients in Africa and attempting a full analysis of plantar ulcer. They pointed out that the sensory state of the foot is not always so anaesthetic as was thought.

Aetiological Studies

These did not begin until 1944, when SILVEIRA wrote as follows: "The pathogenicity is subordinated to a series of purely theoretical ideas invoked to explain it, namely mechanics, the influence of nervous and vascular lesions, the dyscrasias, and certain intoxications. These alone or together have been held responsible, but we must admit that basically it is difficult to say what exact part each factor plays in pathogenesis". This could almost have been written at the present day. SILVEIRA concludes that the major factor is mechanical, stresses the importance of rest, and notes that the uncomplicated ulcer will heal in a plaster cast without any other treatment; he also emphasizes frequent re-examination of the patient so as to avoid the recurrence of ulcer. KHAN (1939) made the important observation that plantar ulcers will heal with rest, including rest if the foot is encased in a plaster cast. This was not at once applied to aetiology, but MUIR in 1943 suggested that the provocative factor was the loss of the plantar cushion due to atrophy of the small muscles of the sole. COCHRANE (1947) stressed the relation of trauma to the aetiology. BRAND (1950) supported this, emphasizing sustained pressure and active injury. PRICE (1959) noted that the distribution of plantar ulcers was related more with walking than standing pressures. GUIDA made an early study (1937) on blood-supply but BARNETSON (1950) made the first detailed study, using oscillometry on neural cases. He showed that local blood vessels had not lost dilatability but vasomotor control was defective. He thought that the neurotrophic changes depended on more factors than vasomotor control damage. GOKHALE (1959) studied temperature changes in feet by

blocking the autonomic system and found that vasomotor control is defective in ulcerated feet of leprosy patients. LEITNER (1938) did arteriography and showed that there is an ample blood supply even in advanced cases, and BANG and colleagues (1938) found normal arteriograms in 26 of 34 ulcerated feet. LECHAT and colleagues (1959) repeated this and could establish no correlation with results of cutaneous thermometry and plethysmography. From all these studies there is no evidence that vascular inadequacy has any part to play in the aetiology of plantar ulcer. Workers in diabetes found the same thing (MARTIN, 1953, 1954).

Methods of Treatment

Some of the suggested therapies have been based on the foregoing theories of causation, but many have been empirical or aimed at suppression of local infection. A few have used local application of antileprosy drugs.

Local nonspecific therapy for which success is claimed includes the sulphonamides (A. ATUCHA, 1945; CHORINE, 1943); streptomycine (FITE et al., 1947); chloramphenicol (IYENGAR, 1959); tyrothricin (CABRERA et al., 1948; MOM et al., 1946); mercurochrome (OBERDORFER et al., 1939); iodoform (LANG, 1930); gentian violet (MUIR, 1941); trichloracetic acid (LAURET et al., 1956). Others were the local application of vitamin A (RYRIE, 1939), of madecassol (LANGUILLON, 1949), beef suet (MAYNARD, 1938), placental implants (FONTILLES, 1955; LUONG et al., 1958), and the infusion of barks of certain South American trees (FLORIANI, 1937), sodium dehydrocholate (HERMANS, 1957; LAVIRON, 1955; REZETTE, 1956), Dettol (RYRIE, 1938), Rivanol (DAS, 1940; MEHTA, 1938). The results recorded a monotonous success suggesting ulcers will heal easily.

Local antileprosy treatment. To use a local injection of an antileprosy drug suggests the suspicion that the ulcer is a specific lesion. Hydnocarpus preparations have been injected in and around the ulcers by LOWE et al., 1937; BOUSEFIELD, 1938; BOSE, 1938; COCHRANE, 1940; MUIR, 1943; BROWNE 1959. In the same way sulphone preparations have been used by DHARMENDRA et al, 1955 and FERREIRA, 1957. Leprolin was used by CALDEIRA in 1948. Results reported were uniformly good.

Treatment by Vasomotor Paralysis

Some workers tried to interrupt the tonic effect of the vasomotor control to try to improve the chronicity of the lesion on the supposition that this was due to inadequacy of blood supply. Thus lumbar sympathectomy was tried by GOHEEN in 1933 and GUADAGNINI in 1950, perifemoral sympathectomy by PY et al. in 1929, C. RUIZ et al. in 1931, BLACK, 1933, VIRNICCHIL in 1941, KIRKALDY-WILLIS in 1945. Epineurectomy or nerve blockage was tried by RANADE et al. in 1957 and VISHNEVSKY in 1938. Also, injection intra-

arterially of vasodilators was tried, ergot derivatives by GOKHALE in 1957, LAVIRON et al. in 1958, WATT-MANEY et al. in 1958 and FRITSCHII in 1959. Acetylcholine has been used for the same purpose by GUIDA in 1937, LANGUILLON in 1946, and A. CASTRO in 1940. All this had discordant results.

Treatment by Orthopaedic Methods

It was soon noted that simple protective footwear had no effect on either the healing or the recurrence of plantar ulcer; hence it seems that the cause could not be simple trauma. Attempts were next made to avoid the concentration of pressure on small areas by using moulded insoles so as to spread the weight. The plaster cast is the simplest method, and its value in healing an ulcer was confirmed by KHAN (1939), HAYTHORN THWAITE (1943), SILVEIRA (1944), FISHER (1955), NEWMAN et al. (1955), BOSE (1956), and LANGUILLON (1960). With a boot of Unna paste GENU et al. obtained similar good results. The plaster boot method for healing the ulcer is the simplest and best, and the patient can remain ambulant. Most cases heal in 4 to 6 weeks.

Treatment by Surgical Methods

Formerly most surgical methods were directed at the chronic bone infection. MILROY (1936) advised it, and others, and many have tried metatarsectomy. The general feeling is that the surgical mutilation does not solve the problem of healing or recurrence. SILVEIRA at the Havana Congress in 1948 protested that "amputation has become a frequent initial treatment of plantar ulcer" and recommended rest and plaster casts.

The Problem of Recurrence

The prevention of recurrence of plantar ulcer is the real acid test of any method or theory of treatment. So far no method of local therapy or surgical intervention successfully meets the difficulty. Why does the ulcer heal in a plaster cast even though the foot carries added weight? BRAND thought it was due to the spreading of the weight over a wide area of the sole by the moulding of the plaster, and he planned moulded insoles of plastic. ROBERTSON (1956) tried the same with moulded leather, also DREISBACH (1959) followed the same line. PRICE (1959) however suggested the interruption of the walking roll was the important factor, and that any type of rigid sole footwear, even wooden clogs, would be beneficial. Soft insoles can be used in addition to the rigid sole. Work is continuing on these lines.

Discussion

It will be seen that the plantar ulcer of leprosy poses three problems:

1. What is the neuropathic lesion responsible for the occurrence of ulceration?
2. Why does the ulcer occur in some neuropathic feet and not in others?
3. Why does the ulcer recur so readily after healing?

The *neuropathic lesion* underlying the lesion has not been the subject of any published study. Some workers are, however, investigating the matter. WEDDELL (WHO, 1960) reported on this and stated that as many as 25% of the sensory fibres to the skin may be destroyed before any sensory deficit can be detected clinically.

It has been generally believed (and stated) that a foot with an ulcer must be anesthetic to pain—and so exposed to unrecognised injury. Recent reports, notably by LAMBILLON (1960) have stressed that this is not in fact the case and that not more than half the cases clinically show this degree of anesthesia. It is also generally stated that there is no loss of deep sensation including joint sense. All these matters need further careful study, and are by no means clarified. It would be of considerable value to be able to recognise by some simple clinical test, which feet are likely to ulcerate and which not. A contribution to this is the recognition of the "pre-ulcerative state" by PRICE (1960).

The *reason for ulceration* in a foot already neuropathic is the second problem. The various theories have been examined in the preceding review:

(i) "Ulcer is due to unrecognised external trauma".
This theory fails because protection of the foot by footwear does not, in fact, prevent ulceration.

(ii) "Ulcer is due to prolonged standing".
This suggestion is untenable because ulcers will heal with the addition to the leg of the weight of a plaster cast, and the added immobility it entails.

(iii) "Ulcer is a specific leprosy lesion".
This view is untenable because of the occurrence of the lesion in diabetes, tabes etc. Nevertheless, as recently as 1959, a paper has appeared describing the use of locally injected anti-leprosy drugs.

(iv) "Ulcer is due to loss of the plantar pad of intrinsic muscles through atrophy".
This suggestion is abandoned because treatment of the foot by using a padded sole had no effect.

(v) "Ulcer is due to prominence of a bony projection in the sole".
This led to metatarsectomy, which is still widely practised. It is significant that no paper has been published stating the value and results of this, but most of those who use the method admit that the ulcer may not heal, may heal and then recur, or may heal and another occur elsewhere on the sole.

(vi) "Ulcer is an expression of local nutritional defect".
This is the reason for the numerous attempts to increase local blood-supply, using chemical or surgical interventions. The results have been reviewed and are not consistently favourable.

(vii) "Ulcer is an expression of deep plantar damage caused by walking".
This recent hypothesis is based on the observations of the effects of walking plaster casts. It should be confirmed by other workers, and may suggest a means of avoiding and treating ulceration.

The *problem of recurrence* has defeated all efforts at solution until recently. All methods of foot-protection, of local therapy, and of locally increased blood-supply have failed to affect the tendency of ulcers to recur.

The recent observations of the effect of plaster casts does, however, suggest an effective method of preventing recurrence of ulceration. It is based on the deduction that as the ulcer has healed while the cast was in position, it would remain healed if the cast was left on the limb indefinitely. As this is not practical, efforts are being made to define the factor that was responsible for initial healing and would, therefore, in all probability, maintain healing. Several workers are following up this suggestion, which appears likely to bring fruitful results.

Conclusion

The recent Conference at Vellore (WHO, 1960) accepted the statement that: "If the present state of knowledge is properly applied, plantar ulceration need not occur in leprosy".

THE LITERATURE ON PLANTAR ULCERS IN LEPROSY 1890–1960

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