Current Practice

MEDICINE IN THE TROPICS

Tropical Ulcers

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Tropical ulcers, cancrum oris, and Vincent's angina are related conditions affecting the limbs, the cheek, and the pharynx respectively. The infecting organisms are identical, though they have been called by a variety of names: the spirochaete has been called *Borrelia vincentii*, *Treponema vincenti* and *Spirochaeta schaudinni*, and the bacillus *Fusobacterium plauti-vincenti* and *F. fusiformis*.

The high incidence of tropical ulcers in tropical and sub-tropical countries is related more to the poor standards of hygiene and medical facilities and the prevalence of concomitant diseases and deficiency diseases than to the tropical climate itself. It is therefore not surprising that these ulcers are seen under similar conditions in temperate climates.

The true incidence of tropical ulcers in tropical countries is not known but it is undoubtedly high. Hospital records in some of these countries have recorded incidences of between 13 and 33% among all new patients registered.¹⁻³ These rates do not include a substantial proportion of patients who are generally content to treat their ulcers at home with native drugs and herbal preparations.

Though frequently small, the ulcers may reach enormous size and cause considerable morbidity and loss of earnings. Even the small ulcers are always a nuisance to the patients, and notwithstanding their size sometimes cause death from complications such as tetanus and gas gangrene. Malignant transformation may occur in chronic ulcers.

Aetiology

Tropical ulcers are caused by a combination of factors. Minor trauma, sometimes too minor to be remembered by the patient, appears to be important. A high proportion of patients (75%) will recall local injury preceding the onset of an ulcer. In others insect bites or stings may lead to the skin being damaged by scratching. The importance of trauma is shown by the fact that the majority of ulcers occur below the knee, usually around the ankles, feet, and toes, at points that are liable to injuries at play on hard ground or walking through bush paths without adequate protection for the feet. These ulcers are also occasionally seen in the upper limbs after injuries.

Neglect or inadequate treatment of minor injuries is the second most important predisposing factor. The application of various native drugs heavily contaminated with faecal matter, or drugs that cause tissue necrosis, results in the devitalization of the tissues and favours invasion by the causative organisms of tropical ulcer, *S. schaudinni* and *F. fusiformis*. Though these organisms abound in large numbers in the ulcer, their source remains in dispute. They are known to be found in the mouth around carious teeth, and it is likely that they are introduced into the cuts by the patients themselves. A study of patients with tropical ulcers showed that 32% carried the organisms in

their own throats.² The not uncommon habit of applying saliva as first-aid treatment to minor cuts is a possible method by which this self-inoculation occurs. It is also possible under the hot humid conditions of the tropics for the organisms to be transferred by flies from one ulcer to a fresh cut in another patient.

Other organisms usually present include *Proteus*, *Pseudomonas*, staphylococci, haemolytic streptococci, and diphtheroids; these represent contamination of the wound, either accidentally or as a result of treatment.

The part played by malnutrition and avitaminosis in the aetiology of tropical ulcers is undecided. These ulcers have been seen in fit persons with no clinical evidence of deficiencies, but it is more than probable that the widespread deficiencies seen in tropical countries may contribute to ulcer formation by lowering the resistance of tissue to infective processes. Vitamin deficiencies were known to be responsible for "trench mouth," an ulcerative lesion of the mouth and pharynx seen among the soldiers of the first world war, in which organisms similar to those causing tropical ulcers were implicated. It is, perhaps, also significant that cancrum oris—caused by the same organisms—is also common among malnourished children in those countries in which tropical ulcers are found.

Clinical Features

The onset and development of acute ulcer to its full clinical extent occur fairly rapidly and are usually complete in about a week. The lesion starts as a blister containing sero-sanguineous fluid, which ruptures in the course of a day to expose an ash-grey, foul-smelling moist slough. The sloughing process may extend rapidly in all directions to involve the skin, the subcutaneous tissue, the muscles, the tendon, and even bone. It may leave an ulcer which is round or oval in shape (Fig. 1) and which may measure from 1 to 40 cm. in diameter, or it may encircle the whole limb. After a few days the centre

Fig. 1.—Acute tropical ulcer. Note the slough involving the extension tendon. (By kind permission of the Editor, *West African Medical Journal*.)

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of the slough begins to liquefy and to separate, exposing a greyish brown base. At this stage the ulcer has a slightly raised edge with considerable oedema of the surrounding tissues and some blistering of the skin beyond its edge. Regional lymph node enlargement, fever, and other systemic signs may be present from secondary infection in the ulcer.

In the chronic tropical ulcer the local and systemic signs seen in the acute ulcer are either absent or slight. The ulcer generally has a hard rigid border and its floor is covered by rather unhealthy pinkish granulation tissue (Fig. 2). The surrounding skin is usually either hyperpigmented or has become thin, atrophic, and depigmented. There is also considerable fibrosis in and around the ulcer. Without the history of the acute ulcer it would be difficult, on inspection alone, to distinguish a chronic tropical ulcer from other types of chronic ulceration in the limb.

FIG. 2.—Chronic tropical ulcer. Note the extensive fibrosis and the area of depigmentation around the ulcer. The ulcer had failed to heal for three years.

Diagnosis

The diagnosis of the *acute* ulcer is a relatively simple matter based on the history, the appearance of the ulcer, and the isolation of the specific organisms from the ulcer base. These organisms can be seen if a drop of fluid aspirated from the floor of the ulcer is examined under a dark ground illumination for the spirochaetes, which will be seen to be motile. They can also be demonstrated if smears of the ulcer fluid are dried and stained with Gram's stain or with 1% carbol fuchsin or gentian violet. The *S. schaudinni* and *F. fusiformis* do not stain very well but their general appearance can be seen, as shown in Fig. 3. The spirochaete measures 7–18 microns, and, unlike other spirochaetes, has loose spirals in it. The bacillus is cigar-shaped, measures 5–14 microns, is slightly curved, and has a beaded or banded appearance due to poorly staining bands. If the services

FIG. 3.—Diagram of *S. schaudinni* and *F. fusiformis* isolated from an acute tropical ulcer.

of a microbiological laboratory are available it is essential that the secondary organisms present in these ulcers and their sensitivities to antibiotics be determined. Examination of throat swabs may also yield Vincent's organisms and other pathogens.

In contrast to the acute ulcers, the diagnosis of *chronic* tropical ulcer is based partly on the history and partly on the exclusion of other chronic ulcerative conditions of the limb. The specific organisms present in the acute ulcer may or may not be present in the chronic ulcer, and other types of chronic ulceration may occasionally be contaminated by Vincent's organisms. It is therefore recommended that all chronic ulcers should be biopsied. The following conditions can be excluded by biopsy and other investigations:

Yaws.—The late ulcerating granulomata of yaws, in which the treponemata are rarely found, can be distinguished from tropical ulcers by a determination of the Wassermann reaction or Kahn test. These tests are positive in yaws but not in a tropical ulcer. X-ray of the bone will show a destructive lesion of the cortex, whereas in a chronic tropical ulcer there is usually a periosteitis with new bone formation.

Veld Sore.—These ulcers, common under desert conditions, have undermined edges and are lined by a grey-coloured debris. The *Corynebacterium diphtheriae* is usually isolated from these ulcers, which have been known to be the cause of paralysis and on occasion to be associated with faucial diphtheria.

Buruli Ulcers.—These ulcers, which are known to be endemic in parts of Australia, Uganda, the Congo, and Eastern Nigeria, are caused by a mycobacterium and may be multiple. The infecting organism, the *Mycobacterium ulcerans*, causes an extensive coagulating necrosis of the subcutaneous fat which then progresses to slough formation. Colonies of the acid-fast organism are present in the centre of the lesion and can be demonstrated by the Ziehl-Neelsen stain. As the slough is cast off it exposes the chronic lesion, which on section contains chronic granulomatous cells with giant cell systems. Healing after about a year is by fibrosis with extensive scar formation.

Varicose or Gravitational Ulcers.—These ulcers, which are associated with incompetent perforating veins around the ankle, may be infected with the Vincent's organisms and present diagnostic difficulties. However, a careful history and an examination of the limb will usually reveal evidence of varicose veins or deep-vein thrombosis preceding the onset of the ulcer.

Squamous-cell Carcinoma usually complicates long-standing chronic tropical ulcers but may also arise without a preceding benign ulcer. The rolled everted infiltrative edge of the ulcer, the destructive lesion of the underlying bone on x-ray, and the presence of regional lymph node metastases are diagnostic. A biopsy of the ulcer clinches the diagnosis.

Malignant Melanoma.—The misdiagnosis of a malignant melanoma as a chronic tropical ulcer is occasionally possible if there is a great deal of dirt or "native treatment" on the wound. A thorough cleansing will generally reveal the true cause of the staining of the ulcer, which should be confirmed by biopsy.

Treatment

The introduction of antibiotics has completely revolutionized the treatment of the acute tropical ulcer. The use of strong antiseptic solutions and preparations containing formaldehyde, popular in the pre-antibiotic era, can no longer be justified. Indeed, such treatment may cause further tissue damage and extend the ulcer. Wherever possible, patients should be admitted into hospital so that the affected limb can be rested completely. After a swab has been taken for culture of the flora and sensitivity tests the patient should be started on a course of antibiotic treatment. Penicillin, 1 mega unit, and streptomycin, 1 g., daily have proved effective and should be continued for about seven to ten days. A change of antibiotics is rarely necessary but may be indicated by the sensitivity of

the secondary invaders. Local treatment of the ulcer should be with gauze soaked in eusol or in normal saline. Stronger antiseptic should be avoided. If the ulcer seems unusually necrotic and dirty a thrice-daily application of hydrogen peroxide for three days should clean this up considerably, so that bland dressings can be resumed. On this regimen the ulcer generally cleans up very quickly and becomes a pink granulating surface in about 10 to 14 days. Small ulcers 5 cm. or less in diameter do not require any skin grafting and should normally heal in about another fortnight. Larger ulcers should be skin-grafted with split-skin grafts taken from an adjacent limb. This may be applied as a single sheet or as a postage-stamp graft. Under less sophisticated conditions pinch grafts can be applied. Before the skin grafting minor surface infection can be eliminated with antibiotic cream applied to the wound for a couple of days. Silver nitrate $\frac{1}{2}\%$ will achieve the same result in some cases.

Where hospitalization is difficult for the patient penicillin can be given as procaine penicillin once or twice a week or as long-acting penicillin once a week combined with oral sulphonamides. The ulcers should be treated locally with eusol gauze changed at least once a day or as often as is both convenient and practical. Gauze dressings soaked in a $\frac{1}{4}\%$ silver nitrate and applied every four hours provide very satisfactory antiseptics without any damage to the epithelium. As soon as healthy clean granulations have formed, paraffin gauze dressings should be applied with firm pressure bandaging, the whole limb being encased in a plaster-of-Paris cast. This will reduce the attendance of the patient at the ulcer clinic, and, by eliminating the frequent changes of dressings, allow the epithelium to grow over the ulcer surface. It is recommended that patients with ulcers requiring skin grafting should wherever possible be admitted into hospital for the purpose, otherwise the grafted limb may be used for normal activity, including playing in a football match.

Irregular attendance or frequent changes of treatment are among factors contributing to the chronicity of tropical ulcers. The use of old-fashioned antiseptic solutions and agents such as acriflavine, which damage the delicate epithelium, cause delay in healing. In time considerable fibrosis occurs in and around the ulcer, which in turn reduces the blood supply of the area and leads to the formation of an unhealthy granulation tissue. Other ulcers become chronic because of an underlying chronic osteomyelitis or the presence of necrotic tendon in the floor of the ulcer. Patients with such lesions should be admitted and a wide excision of the infected bone and tendon carried out. The defect can be covered by either a split skin graft or—where the facilities and resources exist—by a full-thickness pedicle graft.

TODAY'S DRUGS

With the help of expert contributors we publish below notes on a selection of drugs in current use.

Bethanidine

This drug is marketed by Burroughs Wellcome and Co. under the name Esbatal. It is a powerful adrenergic neurone-blocking drug which resembles guanethidine chemically and pharmacologically.

Pharmacology

The first adrenergic neurone-blocking drug to achieve wide use was bretylium, a quaternary ammonium compound. Bretylium was discarded because of irregular intestinal absorption and the rapid onset of drug tolerance which made even

Where there is anaemia or vitamin deficiencies, these should be corrected.

Complications

The treatment of complications of tropical ulcers should be along standard lines. Tetanus and gas gangrene should be treated with large doses of the specific antisera, together with large doses of antibiotics. The greater risk of tetanus and gas gangrene in tropical countries and the absence of facilities for their treatment should emphasize the need for prevention. The enforced attendance of the patient with a tropical ulcer at the ulcer clinic should therefore be utilized to give immunization with tetanus toxoid in addition to the prophylactic coverage with A.T.S. and antibiotic therapy.

Chronic lymphoedema is often seen as a complication of long-standing ulcers. It arises partly as a result of the constriction by the scar tissue of the ulcer of lymphatics on their way from the limb below the ulcer to the groin and partly as a result of chronic infection in the ulcer, leading to a chronic lymphangitis and lymphadenitis.

Malignant transformation of ulcers should be treated along standard surgical lines by a local excision or amputation, combined with block dissection of the regional lymph nodes wherever this is indicated.

Conclusion

Tropical ulcer is a preventable disease and its incidence should decrease significantly with a general improvement in the education, health, sanitation, and economic and nutritional status of the vast majority of people living in tropical climates.

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REFERENCES

- ¹ Burnie, R. M., *W. Afr. med. J.*, 1931, 4, 77.
- ² Ngu, V. A., *ibid.*, 1960, 9, 247.
- ³ Obrien, H. D., *Brit. med. J.*, 1951, 2, 1544.

"Child Care."—This book is composed of articles originally published in the Current Practice section and since revised by their authors. Copies are now available from the Publishing Manager, British Medical Journal, B.M.A. House, Tavistock Square, London W.C.1. Price 30s., postage 2s. 6d.

control of blood pressure difficult. Guanethidine, which was introduced shortly after bretylium, did not suffer from these disadvantages. Bethanidine (*N*-benzyl-*N'*-*N''*-dimethylguanidine) was introduced by the same team of scientists responsible for the discovery of bretylium. Though it resembles guanethidine in many respects bethanidine has several small but real advantages.

In animals an intravenous dose of bethanidine causes a short-lived pressor response followed by a rapid and persistent depression of cardiovascular reflexes. The response of the animal to adrenaline and noradrenaline is increased but that to tyramine decreased. The main action appears to be inhibition of release of noradrenaline at sympathetic nerve endings, and there is no depletion of noradrenaline in the nerves at this stage. After several months the noradrenaline content of tissues does fall.¹

The action of bethanidine in man is similar. It blocks reflexes which depend upon the integrity of the sympathetic vasomotor