TROPICAL ULCER IN NATAL

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The aetiology of tropical ulcer remains obscure and is still a matter of debate.[1-4] This suggests that either a multiplicity of factors are at play or the true causative agent is unknown.

Two types of ulcer are described clinically—the acute and the chronic ulcer—with intermediate types between.[5] The general characteristics of these ulcers would appear to be similar in all parts of the world, but detailed features and manifestations would appear to differ somewhat in different regions. This has been the experience of one of us (J.H.J.), who as a result suspected that an unrecognized aetiological infective agent might exist and be operative, at least in certain cases.

Aetiology

The aetiological factors that have been listed include the following:

1. *Vincent's organisms.*[1, 2, 4, 7, 8] The *Spirochaeta schaudinni* or *vincenti* and the *Bacillus fusiformis* are associated with the ulcers and are invariably reported in any investigation. These organisms are present in some, but not all, of the ulcers examined in some investigations.[6] They are present in small numbers in the mouths of many normal individuals,[9-12] and they are the accepted aetiological agents of infective gingivitis.[9, 11-13] There is evidence to suggest that these two forms, the spirochaete and the bacillus, represent different life stages of the same organism.[4, 9, 11, 13] This, however, is disputed.[11] It is stated that the virulence of the organisms responsible for tropical ulcer is increased by transmission amongst susceptible people.[2] Both the spirochaete and the *B. fusiformis* are obligate anaerobes.[4, 9, 13] Some consider that these organisms are of secondary importance in the lesions.[11, 13, 14] Practically always other bacteria are found associated with them in the lesions.[14] It may well be that they are secondary invaders, the primary aetiological agent being some other organism, e.g. a streptococcus or a staphylococcus, whose virulence could likewise be increased by passage through susceptible people. This view is not accepted by some authorities, and Vincent's organisms are regarded as primarily infective agents in their own right.[15]

2. *Nutritional deficiency.* Deficiencies of various nutritional factors have been considered by different investigators to underlie and promote the incidence of these ulcers. Thus, deficiencies of protein,[3, 16] the vitamin-B complex,[2, 3] vitamin A,[16, 17, 19] essential fatty acids,[16, 17] and calcium,[1, 18] have been postulated. It is likely that a nutritional deficiency is a potent predisposing factor in many non-European communities in Africa and elsewhere. A multiple deficiency is likely to be operative in many situations and cases.[16] In the light of modern knowledge of the non-European diet in Africa the main deficiency at play is one of good-quality protein. Only where extreme dietary deficiencies exist are extensive phagedaenic ulcerative lesions likely to occur.[7]

3. *Debility* due to other diseases and infestations. Examples are chronic malaria, bilharziasis, hookworm, and ascariasis.[2, 3] Such debilitating influences probably operate indirectly by producing a secondary malnutritional state.

4. *Climatic factors.* The consensus of opinion is that a

high incidence of these ulcers is associated with warm moist climates,[1, 20, 21] but the distribution of tropical ulcer is not limited to such regions.[16, 20] The high incidence of tropical ulcer in warm moist climates may be due to a number of factors, which are associated with climate, but may operate independently of it. It would appear to us that the greater incidence in such climates may be related to the improved conditions for survival of the organisms responsible for the condition, particularly with reference to conditions on the skin of the exposed individual; a warm moist state of the skin possibly aiding their survival and facilitating the establishment of infection.

5. *Local trauma.* Most authorities hold the opinion that local trauma is an important aetiological factor in the establishment of this type of ulcer, and that prompt treatment and dressing of such injuries greatly reduces the incidence of ulcers in any labour force or community.[6, 16, 17] But trauma is not essential, for in some cases the ulcer appears to develop on undamaged skin.[2, 19] Trauma would appear to facilitate the establishment of infection by devitalizing the tissues.[15] Devitalization of the skin tissues, whether due to external agency or to endogenous factors, would appear to be a predisposing factor in tropical ulcer.

Epidemic Incidence

The disease takes on epidemic characteristics on occasions.[3, 16, 21] Epidemic outbreaks of tropical ulcer may be due to the following factors:

1. The development of increased virulence in Vincent's organisms due to passage amongst the affected community either (*a*) from ulcer to ulcer or (*b*) through some other bodily focus of infection, e.g. gingivitis.
2. The introduction into a community of virulent Vincent's organisms.
3. The appearance in a community of some other organism normally associated with the ulcers, which either has an inherent high virulence or has acquired a high virulence by passage; this organism producing a primary lesion which becomes secondarily infected by Vincent's organisms, e.g. a streptococcus or staphylococcus.
4. An alteration in climatic conditions which promotes the increased survival, invasion and transmission of the causative organisms.[20] This may be of considerable importance in those areas where sporadic outbreaks of the disease occur, and where large numbers of cases are not a normal medical feature of the district.
5. The exacerbation throughout a community of debilitating factors, e.g. malaria, worm infestation, and deterioration of diet and nutritional status.[16]
6. The appearance in a community of an unknown effective primary infective agent so far unrecognized.

The type of ulceration seen in the tropics raises doubts about the accepted bacterial aetiology of this type of ulceration. It is common to see extensive, deeply penetrating and destructive ulcers, involving muscles, tendons, bones and joints. The granulations are pale, greyish-pink, and gelatinous looking. The involved structures, e.g. tendons, appear oedematous and gelatinous. The slowly progressive and destructive nature of these lesions gives the impression of malignancy, for they frequently do not respond to orthodox treatment and resort to surgery is necessary to produce improvement. Amputation of a foot or leg is sometimes required to save the individual unnecessary misery and to give him an active and healthy, though handicapped, existence. When treating these cases surgically by excision and later skin grafting it is essential to remove completely all the unhealthy gelatinous granulations and to use some antiseptic dressing like BIPP for 1—2 days in order to obtain healthy granulations for the skin grafting. Should any unhealthy granulation tissue survive it provides a focus from which spread occurs to the healthy granulations elsewhere until once more the whole ulcer is covered by unhealthy tissue. That these unhealthy granulations and the slow progress of the ulceration are the sequel to a poor and defective response of the individual and his tissues to bacterial causal organisms, possibly of low virulence, is probable. But the peculiar persistence exhibited by the unhealthy granulations in such cases leaves doubt that this explanation is adequate (J.H.J.).

A NATAL EPIDEMIC OF TROPICAL ULCER

In May 1958 it was reported that an epidemic of tropical ulcer was occurring amongst Native school children and farm labourers in Verulam District, Natal. This is a warm district on the North Coast of Natal, just north of Durban. There had been exceptional heavy rains during the previous months, which had been very hot and moist. It was found on investigation at one school that 83 children were suffering from the complaint. The affected labourers were mainly from an estate where it was known that dietary conditions and general housing facilities for them were of a poor standard.

The ulcers in some cases were large and extended through to the subcutaneous tissues, and possibly as far as the muscle sheaths of the limbs. In both children and labourers the ulcers caused incapacity, and in the severer cases marked disability. The worst ulcers were seen in those labourers who were obviously malnourished. In some of the children the ulcers were small and relatively superficial. The granulations of the ulcers were red and healthy looking in most cases. None presented the appearances or had the severity of penetration of those which had been frequently seen in the tropics by one of us (J.H.J.). Some of the children were suffering from lesions of an impetiginous character on the lower limbs.

The two following cases are typical:

Case A

N.N., Native cane-farm labourer aged 15-16 years. Clinically malnourished. Temperature normal.

Appearance of ulcer. Sloughing purulent ulcer on the middle of the anterior surface of the right shin. Indurated swollen margins. Yellow slough on floor of ulcer, which was oozing serum. When the friable slough was detached, healthy red granulations were exposed. Slight inguinal adenitis.

Swab taken from ulcer. (1) Direct smear of pus showed many pus cells, many fusiform bacilli, some spirochaetes, and a few Gram-positive cocci. (2) Aerobic culture on blood agar gave a scanty growth of β haemolytic streptococci and diphtheroids. (3) Culture in Robertson's chopped-meat medium gave a heavy growth of streptococci, in which some fusiform bacilli were present.

Biopsy of ulcer. Sections show an acute and chronic ulcer with an undermined edge. Acute and chronic granulation tissue is spreading deeply in the dermis. Masses of fusiform bacilli and a few spirochaetes are present. Some cocci are also seen.

No stool or blood specimens were obtained.

Case B

J., Native cane-farm labourer aged 15-16 years. Clinically malnourished. Temperature normal.

Appearance of ulcer. Sloughing purulent ulcer on the anterior surface of the upper right shin. Indurated raised margins. Pus not so abundant as in case A, and ulcer less septic looking and more chronic in appearance. Red healthy granulations. Slight inguinal adenitis.

Swab taken from ulcer. (1) Direct smear of pus showed many pus cells, many fusiform bacilli, some spirochaetes and a few Gram-positive cocci. (2) Aerobic culture on blood agar gave a scanty growth of β haemolytic streptococci and diphtheroids. (3) Culture in Robertson's chopped-meat medium gave a growth of fusiform bacilli and some streptococci.

Biopsy of ulcer. Sections show an acute and chronic ulcer with an undermined edge. Acute and chronic granulation tissue is spreading deeply in the dermis and into the subcutaneous fat. Masses of fusiform bacilli, some spirochaetes, and many mixed bacteria, mainly Gram-positive cocci, are seen.

No stool or blood specimens were obtained.

Table I gives the results of investigations carried out on some of the children exhibiting ulcers at one school.

Samples of earth and dust from the school playground were collected for bacterial examination. The following organisms were found and isolated:

Fungi morphologically resembling *Geotrichum*, Gram-positive cocci, Gram-positive bacilli with terminal spores resembling *Clostridium tetani*, Gram-positive bacilli resembling diphtheroid bacilli, haemolytic lactose-fermenting coliform bacilli, *Bacillus subtilis*, staphylococci.

It will be seen from Table I and from cases A and B, as demonstrated by direct smears or on culture from the swabs taken from them, that a multiplicity of organisms were associated with the ulcers. The following organisms were found: (1) Fusiform bacilli, (2) spirochaetes, (3) streptococci (α haemolytic streptococci and, the most frequent, β haemolytic streptococci), (4) coagulase-positive staphylococci, (5) Gram-positive bacilli (mainly diphtheroids and *Bacillus subtilis*), (6) Gram-negative bacilli (mainly coliform organisms and *Ps. pyocyanea*), (7) yeasts.

Vincent's organisms occurred in 50% of ulcers examined, streptococci in 80%, and staphylococci in 60%.

TABLE I. INVESTIGATIONS ON SCHOOLCHILDREN WITH ULCERS

Case No.	Initials Age Sex	Ulcer Swab			Gums		Stool	Blood	Remarks
		Direct Smear	Aerobic Culture on Blood Agar	Culture on Robertson's Chopped Meat	Appearances	Swab			
1	E.M. 11 yrs. M	Pus cells. Gram + cocc. Gram − bac.	*Ps. pyocyanea*	Gram − bac. Fusiform bac. Streptococci	Apparently normal gums	Vincent's organisms*	Ova of AL, TT, SH	WR −. Eosino. 9%	
2	M.D. 12 yrs. F	Pus cells. Few yeasts	Coagulase + staphylococci	Mixed staphylococci and streptococci	Apparently normal gums	Vincent's organisms.* Haemolytic streptococci on culture	AL	WR − Eosino. 1%	
3	R.Z. 14 yrs. M	Pus cells. Gram + bac. Gram + cocc.	β haemolytic streptococci. Coagulase + staphylococci	Mixed staphylococci and streptococci	Bleeding spongy gums	No Vincent's organisms seen	Ankylostoma. AL, TT	WR doubtful. Eosino.?	
4	R.M. 10 yrs. M	Pus cells. Gram + cocc. Some yeasts	Mixed coagulase + staphylococci and *B. subtilis*	Mixed staphylococci, streptococci and *B. subtilis*	Apparently normal gums	No Vincent's organisms seen. Haemolytic streptococci on culture	TT	WR −. Eosino. 6%	
5	F.M. 16 yrs. F	Pus cells. Fusiform bac. Spirochaetes. Gram + cocc.	β haemolytic streptococci	Streptococci. Fusiform bac.	Apparently normal gums	Vincent's organisms.* Haemolytic streptococci on culture	Cyst EC, TT	WR −. Eosino.?	Typical ulcer on right shin. Slight inguinal adenitis.
6	W.K. 13 yrs. F	Pus cells. Gram + cocc. Tiny Gram − bacilli	Coagulase + staphylococci	Staphylococci	Apparently normal gums	Vincent's organisms.* Haemolytic streptococci on culture	AL	WR −. Eosino. 2%	
7	S.G. 12 yrs. M	Pus cells. Red cells. Gram + cocc.	β haemolytic streptococci		Spongy gums	Vincent's organisms*	AL	WR −. Eosino. 11%	Typical ulcers—one due to secondary spread from original ulcer, which was healing.
8	M.N. 10 yrs. M	Pus cells. Fusiform bac. Spirochaetes. Gram + cocc.	β haemolytic streptococci. Coagulase + staphylococci	Mixed streptococci & fusiform bac. Some spirochaetes on dark-ground illumination	Apparently normal gums	Vincent's organisms*	AL, TT	WR −. Eosino. 26%	Early ulcer over right ankle below internal malleolus, indurated area due to cellulitis around it. Superficial skin detached and undermined. Inguinal adenitis.
9	A.D. 9 yrs. M	Pus cells. Fusiform bac. Gram + cocc.	α haemolytic streptococci. Coliform organisms	Streptococci. Fusiform bac. Gram − bac. Some spirochaetes on dark-ground illumination	Bleeding spongy gums	Vincent's organisms*	No ova or cysts found	WR −. Eosino. 3%	Early ulcer with crust and undermined edges, oozing serum.
10	H.M. 9 yrs. M	Pus cells. Fusiform bac. Gram + cocc.	Coagulase + staphylococci	Staphylococci. Gram + bac.	Absent and not available for examination		AL	WR −. Eosino.?	Early ulcer back of right heel over tendo achillis. Scabbed, oozing serum, red granulations.

* i.e., fusiform bacilli and spirochaetes.
cocc. = cocci. bac. = bacilli. AL = *Ascaris lumbricoides*. TT = *Trichocephalus trichiura*. SH = *Schistosoma haematobium*. EC = *Entamoeba coli*. WR = Wassermann reaction. Eosino. = eosinophils.

Intestinal parasites were present in 90% of the cases.

Malnutrition as shown by spongy and bleeding gums was obvious in 30% of the cases.

Swabs taken from the gums showed Vincent's organisms to be present in 73% of those examined, and haemolytic streptococci in 40%.

No evidence was obtained to indicate a previously unrecognized aetiological agent for the ulcers.

Treatment Adopted to Deal with the Outbreak (A.B.)

A total of 86 school children were treated. Each child was given 300,000 units of procaine penicillin G intramuscularly at the outset of treatment. The ulcer or ulcers were dressed daily with eusol for 1 week. A further injection of penicillin was then given, and an elastoplast adhesive bandage was firmly applied from foot to knee and left on for 14 days. When the elastoplast was removed, 75 of the cases had healed completely. The remaining 11 cases were re-strapped with elastoplast for a further 14 days, and at the end of this period only 2 cases were found incompletely healed and required further strapping.

The efficacy of penicillin as a method of treatment has been reported. It would appear to be an effective treatment for the acute and early ulcers which have not reached a stage of true chronicity.[22]

In the use of antibiotics in the treatment of these ulcers, choice of chemotherapy based bacteriological sensitivity tests has been advocated. It has been stated that the bacterial flora varies from ulcer to ulcer and in individual ulcers as they progress through the various stages to chronicity.[23] Antibiotics, as an adjunct to surgery, are invaluable with the large chronic indolent ulcers seen in the tropics.[16, 24]

Discussion

The bacterial findings show several potential infective agents to be present in the ulcers. Of these, streptococci, staphylococci, and Vincent's organisms predominate, in that order. The organisms responsible would appear to have been sensitive to penicillin.

The characteristics of the outbreak and the conditions at the school suggested direct contagion as the method of spread of the infective agent. The children sat close together at long school-desks, often with their legs touching. The ulcers were uncovered or poorly protected by dirty improvised dressings. Direct contagion from ulcer to clean leg was highly probable.

Another means of spread, possibly equally important, was from the mouths of the children to the legs of those afflicted with ulcers, either autogeneously or heterogeneously. Scratching with spittle-contaminated fingers would lead to effective auto-inoculation.

Trauma was likely to have played a part in the onset of some of the ulcers, particularly those located over the malleoli at the ankles.

The hot humid conditions which preceded and accompanied the outbreak may have had an influence in promoting the epidemic.

The high incidence of worm infestation amongst the children, and the evidence of malnutrition, would appear to incriminate both as contributory factors in the incidence of the disease. The children as a whole were considered malnourished. Spongy unhealthy gums are associated with two recognized malnutritional states—scurvy, in which it is well recognized, and pellagra (Alpine scurvy).[25, 26] Both these malnutritional states occur in subclinical forms, not easily recognizable. Both are common amongst the indigenous population of Africa who suffer mainly from a multiple deficiency.[16, 26] Unhealthy gums would promote the survival and spread of infective organisms in the mouths of an affected community and possibly assist in enhancing their virulence. The role of malnutrition may be a dual one in epidemics of tropical ulcer, namely the promotion of a high incidence of pathogenic organism in the mouths of the community and a low general bodily resistance to them.[27] Noma, or cancrum oris, is usually attributed to a similar pathology. It occurs in debilitated or malnourished infants and is rarely seen today. Cases do occur amongst the African community (J.H.J.).

In dealing with an outbreak of tropical ulcer it is reasonable to conclude that oral hygiene should receive attention, especially when institutions and schools, and like communities, are afflicted.

Streptococci and staphylococci were isolated from the ulcers more frequently than Vincent's organisms. The authors feel that the role of the two former organisms may have been that of primary infective agents directly responsible for contagion. Some of the children were suffering from impetiginous lesions on the legs—lesions which are normally associated with streptococci and staphylococci. Virulent streptococci and staphylococci are highly contagious, but are not normally associated with the type of ulceration encountered in the epidemic. The ulceration may have been due to concomitant or superimposed infection with Vincent's organisms on a lesion primarily impetiginous at onset but rapidly modified by the superimposed Vincent's infection.

That the organisms in the mouth are potentially dangerous is borne out by experience. One of us (A.L.F.) recalls the following case which occurred many years ago: A European miner struck a Native labourer on the mouth, cutting the knuckle of his index finger on the labourer's teeth. Within a few days severe sepsis developed in the injury and spread so that hospitalization was necessary. Swabs taken from the exuding pus disclosed an almost pure infection by Vincent's organisms. Swabs from the mouth of the assaulted Native labourer showed Vincent's organisms to be present in his mouth, though his gums were healthy.

The ability of Vincent's organisms to act as a primary infective agent must be accepted when the literature quoted is considered.[28] This is not disputed, but it is considered that their relative infectivity may fluctuate in relation to that of concomitant organisms associated with them, so that in certain epidemic settings the latter may take over the primary contagious role.

SUMMARY

1. The aetiology of tropical ulcer is reviewed.

2. An epidemic of tropical ulcer in Natal is reported and its features discussed. Aetiological factors at play included bacterial infection, malnutrition, parasitic infestation, trauma, and possibly climatic influences.

3. The significance of organisms other than Vincent's in relation to epidemic outbreaks of tropical ulcer is discussed.

4. It is considered that oral conditions, possibly aggravated by malnutrition, may provide a source of contagion and should receive attention in dealing with these ulcers, especially in schools and institutions.

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