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Observations on the Etiology and Treatment of Lichen Planus Atypicus and "Jungle Rot"

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INTRODUCTION

THERE are several types of subacute or chronic inflammatory and ulcerative changes of the skin and mucous membranes of the mouth which medical officers encounter during a prolonged tour of duty with combat troops operating in the tropics. The author is interested in two types of these dermatoses: viz., Lichen planus atypicus and a subacute or chronic serpiginous but relatively painless, eczematous ulceration of the skin which tends to spread rapidly and resist healing, commonly referred to as "jungle rot."

Lichen planus atypicus and "jungle rot" are of particular interest to the writer for the following reasons: (a) Their etiology is not yet definitely established. (b) The two conditions are not yet properly defined nor generally accepted as entities by many men in the profession. (c) They were major causes of military disabilities and evacuations to the Zone of the Interior for troops operating in the tropical climates of the Asiatic-Pacific and African theaters of war during the period 1942 to 1945. (d) The therapy to date is experimental. (e) Relapses and recurrences are common, thus creating problems of postwar management for veterans' facilities and clinicians who will serve these men when they are released from military duties.

This paper represents the conclusions drawn from a clinical laboratory study and management of 100 cases of chronic dermatosis in military personnel operating in New Guinea during the pe-

riod 1944 to 1945. Twenty-one of these cases were diagnosed as Lichen planus atypicus and seventy-nine were diagnosed as the chronic eczematoid and eczematoid-exfoliative types of dermatosis referred to as "jungle rot." During a five and one-half month period of special study, 33 per cent of the cases of a large dermatologic service of a general hospital were due to Lichen planus atypicus and "jungle rot," and 60 per cent of the dermatologic cases that were evacuated to general hospitals in the continental United States were due to these same conditions. Twenty-six per cent of all clinic consultations were for cases coming under these two categories.

LICHEN PLANUS ATYPICUS

This is a relatively new entity because there is no pathognomonic diagnostic bacteriology or serology associated with the disease. There are those who question whether it should be considered a separate entity at all. The facts are, however, that the Lichen planus atypicus of the tropics differs from the typical Lichen planus more frequently encountered in temperate zones. Lichen planus atypicus presents localized atrophic sequelae, does not have typical papules in the early phases and shows a marked tendency to hyperpigmentation along with the lesions. These three differences are supported by minor differences which will be revealed as the discussion of some of the apparent secondary etiologic factors and body fluid changes are given below. The following factors and findings were present in the 21 cases studied in this report: All of the men had been taking 60 mg. (1 grain) of atabrine daily for several months as part of the malaria control program requirements. I believe that atabrine skin sensitivity is a major etiologic factor in Lichen planus atypicus in spite of some of the observations of Bagby[1] to the contrary. All of the men gave histories of excessive exposure to direct sunlight in the tropics; profuse sweating was a common history. All of the men were on overseas combat rations and showed evidence of vitamin B and C deficiencies. Generally the initial lesion seemed to start with some form of physical injury to the skin. An emotional or neurologic factor was pronounced in several cases. Blood serum studies showed a hypoproteinemia in all of them and an albumin/globulin ratio approaching 1:1. Hypocalcemia was present in advanced cases. The blood calcium level averaged 8 to 8.5 mg. per cent.

The clinical course of the disease was as follows: The primary lesions are generally pruritic, reddish or purplish patches on the skin which spread slowly week by week. Later these patches develop scaly, raised areas, followed by atrophic changes and hyperpigmentation. The earlier topographic distribution of the lesions are the extremities, groin, external genitalia and neck. These lesions later appear on several other parts of the body and in the mouth. Their progress is slow, but they are resistant to practically all forms of treatment as well as subject to relapses and recurrences after apparent improvement. Several of the men who have returned from the hot, moist climate of the tropics and have discontinued atabrine therapy have made temporary improvements, but after two to three years some are now showing signs of a relapse.

The treatment of Lichen planus atypicus is nonspecific. It is important to correct the defects listed above by giving a diet high in proteins, vitamins B and C and in calcium. If the patient is in the tropics, he should be sent to a cooler climate if possible and all drugs which are excreted by the skin should be temporarily discontinued. It is also important to control the itching by the use of one of the following prescriptions:

(a) A lotion of — Gm. or Cc.

Phenol	2.0
Glycerine	15.0
Lime Water in sufficient quantity to make	120.0

Directions: Shake well and apply liberally to affected parts twice a day after a bath.

(b) Warm oatmeal or cornstarch tub bath of twenty to thirty minutes duration twice daily until itching is less. Then take the bath each night at bedtime. The bath is prepared by adding one-half to one pound of oatmeal or cornstarch to a tub containing about forty gallons of warm water. This bath is contraindicated in the presence of secondary infections.

Special effort should be made to prevent the traumatization of the local areas. Any form of external trauma will invite resistant secondary infections and cause the lesions to spread more rapidly.

"JUNGLE ROT"

The medical profession does not agree that this is an entity, but the condition which will be described here is so firmly entrenched in the minds of the laymen as "jungle rot" that the profession must consider the disease from the standpoint of pathogenesis and treatment. A more appropriate name for the affection might be chronic eczematoid-exfoliative dermatosis of the tropics, which develops following cutaneous trauma of many types. The etiologic factors in "jungle rot" are numerous and nonspecific. From a study of the 79 cases covered in this report, the factors are as follows:

1. Hot climate with high humidity and a type of activity which causes profuse sweating.
2. A canned ration combat diet with resulting avitaminosis, especially of B and C.
3. Some form of local, physical or chemical skin trauma, such as chigger or insect bites or stings; thorn, splinter or barb pricks; coral bruises; chemical or hot water burns; exacerbation of a previous athlete's foot infection or its reaction, or dermatitis venenata due to contact with the many types of plants in these tropical areas of the Southwest Pacific. The injuries due to direct enemy action such as bullets, bayonet or shrapnel wounds are not included in this study.
4. Neglect of local injury which permitted marked secondary infections. These infectious agents in the presence of wet, dirty clothes potentiated each other and accelerated the spread of the infection. Some of the neglect was due to ignorance and carelessness because the men did not realize the importance of immediate medical care to slight skin trauma under these tropical conditions.
5. The microorganisms isolated and identified in these lesions were: *pyogenic cocci,* dominated by necrotizing staphylococci; *spirochetes and fusiform bacilli* which frequently resemble, both by staining and morphologic characteristics, those seen in cases of Vincent's stomatitis; *Corynebacteria.* Some of these were true diphtheria exotoxin producers as shown by culture, animal inoculation and on epidemiologic grounds, with postdiphtheritic neuritis or myocarditis. Ward and Mason[2] in their study of men with similar ulcers showing a dominance of Corynebacteria in North Africa found that the loss of oculomotor accommodations were the chief manifestation of neuritis following "jungle rot." Fungi: Epidermophyton inguinale, Trichophyton gypseum, Trichophyton interdigitale and Trichophyton purpuerum; Monilia albicans and Monilia candida; Cryptococcus hominis; Oidium pulmoneum and Oidium dermatitidis. Gram-negative rods: In lesions in the regions of the axillae, groins or lower parts of the trunk, members of the Escherichia and Pseudomonas genera were present in a large percentage of cases and in large numbers. Vibrio: Morphologic vibrios were frequently found in these lesions, but they were not identified.

It is quite evident from the multiplicity of microorganisms found in these lesions that there is no single animate, etiologic agent of "jungle rot"; at least such a factor has not yet been isolated and identified. March and Wilson[3] felt that the causation may be summed up by 4 F's: filth, food, friction and fusospirillosis. Several other investigators felt that the three major factors were a hot, moist environment which depresses the physiologic defense of the host, local trauma which does not receive proper and immediate care, and a multiplicity of contaminations with synergistic pathogens.

The investigators of "jungle rot" by laboratory methods in general agree that for moist, hot climates the dominant microorganisms are pyogenic cocci which tend to produce rapidly sloughing, undermined phagedenic ulcers, and the organisms of the Vincent's stomatitis-like complex which add a grayish pseudomembranous appearance to the crater. James[4] found the Vincent's spirochete and the fusiform bacillus the dominant organisms in his study of "jungle rot" lesions in the South Pacific Archipelagos. Berry[5] agreed with James[4] and felt that the fusiform bacillus was more important in the production and proliferation of the ulcer than the Spirochaeta Vincenti. Berry [5] and Costa[8] thought that the reason more investigators have not found the bacillus fusiformis in a large percentage of the ulcers cultured and in larger numbers was due to the fact

that they took superficial swabs only, while the fusiform bacilli are more abundant in the deeper parts of the ulcers. In these moist, hot climates the Corynebacteria infections in the ulcers and their neuritic and myocarditic sequelae are important, but they do not occur in as high a percentage of cases as they do in the hot, dry climates where the Corynebacteria often are the dominant organisms. This is shown from reports such as that of Colonel Liebow and his associates[6] and of Gill[7] who investigated tropical ulceration in North Africa and Palestine. There they were sometimes called ecthyma or tropical ulcers, but their differences from "jungle rot" are chiefly due to the dryness of the environment. Very few investigators have studied in detail the mycologic factors in "jungle rot."

Except in cases where the Corynebacteria were present, the patient showed little or no general symptoms or physical signs. The local lesions were the chief and only complaints. A more detailed study of the local ulcerations will frequently reveal acanthotic changes, edema of the corium and infiltration of lymphocytes and plasma cells into that layer. In more chronic ulcerations pseudoepitheliomatous hyperplastic changes may be present which resemble epidermoid carcinoma, while dense fibrosis on a base of granulation tissue replaces the edema of the corium more commonly seen early in the disease. In a hot, humid environment these ulcers can spread rapidly from a minute papule caused by a chigger attachment to an ulcer of 3 to 5 cm. in diameter which involves the muscles and/or bones within two weeks. The red blood cell count in cases of "jungle rot" shows no significant changes. The white blood corpuscle count shows only changes related to the dominant agent of the secondary infection and the fulminating state of the disease.

TREATMENT

Just as the causes of "jungle rot" are multiple, the treatment is nonspecific and experimental. In the initial stage of the treatment the patient should be placed in a hospital and treated preferably as a bed patient even though he feels well except for the local lesions. The local lesion should not be treated too vigorously, especially where the treatment involves wetting or softening agents such as aqueous solutions or ointments. One should beware of overtreatment! If it is at all possible and the patient is in the tropics, he should be sent to a cooler climate, given a high protein-vitamin B and C diet, or the diet may be supplemented with these vitamins given by mouth or injection.

The methods which the author used in the management of these 79 cases were:

(a) Penicillin-triple typhoid injections—A total of 300,000 to one million units of penicillin G were given in divided doses of 25,000 to 50,000 units each intramuscularly every 3 hours night and day until the total dose was given, depending on the temperature reaction, pulse pressure and pulse rate. These reactions were chiefly the results of the triple typhoid vaccine which was given each patient along with the first injection of penicillin or between the first and second injections. The standard triple typhoid vaccine was given for the purpose of producing a form of shock fever therapy. It was given in 0.2 cc. amounts intravenously, generally mixed with 500 to 1,000 cc. of isotonic saline solution. If the patient developed a temperature of 102° F. or above or had a chill, the standard triple typhoid vaccine injection was not repeated. If, on the other hand, no such temperature or shock response occurred from the first typhoid vaccine injection, a second intravenous injection of 0.2 cc. of the same vaccine was given three hours after the first. Most of the patients responded well with fever and one or more chills from the first injection of vaccine. None were given more than two injections even though a small percentage did not show a fever or a chill response. These nonreactors nevertheless received the full series of penicillin G injections.

The results of this drastic type of therapy were dramatic. In 74 per cent of the cases the ulcers of six to eight months duration showed healthy bases and granulations in forty-eight hours and were healed within ten days to three weeks. Twenty-six per cent of the cases did not respond any better to this drastic treatment than to some of the milder and more prolonged methods which will be described below.

(b) Soak the ulcers in warm potassium permanganate solution, 1 part to 8,000 of water, for twenty minutes three times a day. If there is considerable weeping or fissuring, dilute the solu-

tions to one-half or one-quarter of the strength just given. As the local condition improves, increase the strength to 1 part to 8,000 or even 1 part of potassium permanganate to 3,000 parts of water. Dry the local lesion well after each treatment and leave the lesion open to air, to dry heat under a cradle, or to sunlight for a short period daily. If there are fissures between the toes, as sometimes appear with concomitant athlete's foot infection, between the regular scheduled soaks keep the toes apart with sterile gauze soaked in borax and/or sulfadiazine ointment. If penicillin incorporated in a water-soluble ointment base is preferred to the sulfadiazine, it is to be remembered that such a penicillin ointment must be kept at 10° C. or below when not being used or it will lose 65 to 85 per cent of its potency within one week. If there is an associated reaction present with the athlete's foot infection, one must remember that the primary lesion is the one of greatest importance and symptomatic therapy to allay itching is all that is necessary for the local id lesions. Where secondary infections are severe, a full course of sulfathiazole or sulfadiazine by mouth should be given.

(c) In chronic and less fulminating lesions where a fungus appears to be the dominant organism, the treatment should be as follows: clean the ulcer and apply locally on alternate days a 2 per cent gentian violet solution in 70 per cent alcohol, or try locally the new drug suggested by Kleinberg and Meyers[9], viz., monopyridine iodine p-nitrobenzoate or Zephiran HCl concentrate suggested by Travis.[10]

(d) Where there is some hardness of the edges of the ulcer, use Frazer's solution locally:

Rx	Gm. or Cc.
Salicylic Acid	2.0
Benzoic Acid	2.0
Tincture of Iodine	10.0
Spirit of Camphor in sufficient quantity to make	60.0

(e) Where considerable sloughing is present, the ulcer has overhanging edges and the craters are filled with secretion, the lesions should be cleaned up with mild borax or saline solution, the edges cut back and the following dressing applied on alternate days:

Rx	Gm. or Cc.
Brilliant green	1.0
Crystal violet	1.0
Alcohol (50%) in sufficient quantity to make	100.0

In the lesions where the true exotoxin Corynebacteria dominate, the administration of 10,000 to 20,000 units of diphtheria antitoxin intramuscularly is indicated after testing for serum hypersensitivity. It is also advisable in such cases to apply powdered tyrothricin to the local lesion. The action is due primarily to the polypeptid gramicidin fraction which makes up 20 to 35 per cent of tyrothricin and causes bacteriostasis and bactericidal action by inhibiting the phosphorylation-nutrition cycle of the bacteria. The polypeptid tyrocidin which makes up 60 to 65 per cent of tyrothricin is a strong tissue poison in general and thus does not show the selective antibiotic effect on the bacteria without also harming the host tissue.

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