

NEW SKIN DISEASES IN AFRICA

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Mr. President, Ladies and Gentlemen,

When I speak of new diseases I imply not only diseases recently described for the first time, but also diseases well known in other parts of the world but only lately recognized in Africa. The diseases I have chosen to discuss are some in which studies of the environment have helped or may still help in defining their nature and causes.

The subject can best be introduced by two clichés :

Ex Africa semper aliquid novi : and

There's nothing new under the sun

and a quotation, "A distinctive feature of tropical medicine is that the number of reported cases of a given disorder is not solely dependent on its real incidence, but rather on the interest shown in it" (Camain, 1969).

I start on this note because I know to my cost that when you start practising dermatology in a new environment and with patients whose skin colour is different from the one you are used to you may be misled in various directions.

Diseases common in all countries may be so modified by skin colour as to look quite different on a black and a white background and, particularly in tropical Africa, lesions may, for reasons known or unknown, be much more exuberant and florid than they are in temperate zones. On the other hand, newcomers may notice things of significance missed by older Africa hands because they were accepted as part of the landscape. As Africa is still very largely unexplored from the dermatological point of view there is plenty of scope for finding dermatoses still undescribed anywhere or stated not to occur in the inhabitants of Africa.

Anybody used only to dermatoses of white people who contemplates moving to the tropics of Africa or elsewhere would be well advised first to take six months off and read all the old literature on tropical diseases that he can lay hands on. It is astonishing how much has already been described, sometimes over and over again under a great variety of titles in what Simons described as the jungle of literature on tropical dermatology.

Rediscovery of diseases is all very well provided you can add something new, but a rehash of something that everybody ought to know is pointless. Two recent examples come to mind : somebody in South Africa tells us that nephritis is a complication of secondarily infected scabies, and someone else in the United States has rediscovered *condylomata lata* between the toes. William B. Bean (1967) summed this subject up when he said that the only thing new about the Stevens-Johnson syndrome was that it was news to Stevens and Johnson.

I have made a few rediscoveries in my time, some of which are in print to dash me if I get an attack of hubris. I found when I started working at the London Lock Hospital that the posterior auricular lymph node that sits on the mastoid process is frequently enlarged and the finding is of diagnostic value in secondary syphilis ; this was not noted in my textbooks but I later found that Fournier had described it a century before. When I went to Africa I rediscovered *witkop* (white head) which had been described as an unusual manifestation of syphilis found in Bechuanas, and I did not recognize that it was simply endemic favus in people with endemic syphilis as well.

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I should have done because I had previously seen the same phenomenon in North Africa where no confusion ever existed. A more recent mistake was to think I had found a new disease of elastic tissue; it was simply very extensive post-rhagadic scarring of the face with a histological picture of elastosis instead of the usual elastolysis (Marshall, 1968).

A more interesting rediscovery was suddenly-appearing peladoid alopecia in children following within a few days of a tick bite on the scalp. The hair loss may be temporary or permanent. The ticks had always been removed, but the descriptions given suggest that they were ixodid. The cause is probably either a toxin or anti-coagulants in tick saliva (Marshall, 1966).

SHIN DISEASES

The shins are a site of election for skin diseases in the African Negro and the commonest conditions seen are impetigo, ecthyma, tropical ulcer and infective eczema; the varicose ulcers of Caucasians are extremely rare.

Recently, two new shin diseases have been discussed. Harman (1968) has restudied dermatitis cruris pustulosa et atrophicans (sycosis cruris) first described by Clarke in 1952. The condition bears some resemblance to Bockhart's impetigo, but is very resistant to treatment. Although I have seen it in South Africa and have had reports of it from other parts of Negro Africa it appears to be most prevalent in young adults in Nigeria and the vicinity. Conditions similar or identical to it have also been reported from tropical or subtropical areas of other continents.

Ocreiform atrophy and dermatitis of the shins has been described by Verhagen and Koten (1968). This condition which, to my mind, presents all the hall marks of an infective eczema is common in Kenya. I see such eczemas in Bantu and Coloured patients in South Africa and classify them as infective or nummular eczema.

In neither of these diseases was malnutrition seemingly implicated, but in both it has been suggested that oiling of the skin, with coconut oil or other vegetable oils in Nigeria, and with Vaseline in Kenya, may be a causative or contributory factor. The geographical distribution of the diseases certainly suggests that local environmental factors are involved. But if oiling of the skin is a basic factor it is odd that only the shins are involved, because Negroes seldom confine their oiling ritual to the legs alone. Further, coconut oil and Vaseline are commonly used all over black Africa.

One basic common factor in all the shin diseases is the dry shin phenomenon so often seen in Negroes, but seldom discussed in the literature because it is so commonplace. Piers (1947) of Nairobi, Kenya, mentioned it in an article in which he complains about crazy pavement dermatosis or mosaic lesions of the shins being accepted automatically as signs of pellagra; and Ross (1966) found what he called the crackled leg syndrome in 20 per cent of Venda children in the Northern Transvaal, South Africa.

SCHOOL NO.	SEASON	TOTAL EXAMINED	TOTAL XEROSIS	DRY SHINS %	CRACKLED SHINS %	ECZEMA OR INFECTION %
1	Wet	654	115 (17%)	81	17	2
2	Dry	231	79 (33%)	73	26	1
3	Dry	216	81 (36%)	75	24	1

TABLE I.—Dry shins in Zambia (Dr. Brian Reid, 1969)

Dry shins may be a physiological state in some Negroes, but one must not dismiss malnutrition entirely as it is a standard state in most of Africa. A pilot study of 1,000 school children in Zambia by Brian Reid illustrates not only this but the effect of seasonal changes on the shins (Table I). Reid finds the association of dry shins with low weight for age highly significant statistically and says he is tempted to conclude that during the wet season a hard core of malnourished children will manifest dry shins regardless of environment; and during the dry season the harsher climate will produce xerosis in normal and malnourished children.

To sum up, the factors contributing to tropical shin disease in general are probably these :

1. Dry shins whether physiological or due to malnutrition.
2. Trauma to legs that are bare some or all of the time
 - (a) physical
 - (b) heat from fires ; and possibly wood smoke (Piers, 1947)
 - (c) sun.
3. Irritants, allergens, and perhaps photosensitizers : strong soaps, oils, and medicaments modern or traditional.
4. Bacterial and fungal infections.

As to photosensitizers, I would note that F. P. Scott of Bloemfontein, South Africa, believes that mutton fat, lanoline and Vaseline may act as photosensitizers.

Before leaving the subject let me warn you about pseudo crackled shins. This condition disappears with soap and water as do most cases of "acanthosis nigricans" in the African Negro.

GRANULOMA MULTIFORME—THE MKAR DISEASE

In 1962 I was told by Susanne Kok, a medical missionary working in the leprosy settlement at Mkar, of a chronic disease resembling granuloma annulare that was prevalent among middle-aged and elderly people in the Tiv division of Benue Province in Northern Nigeria. Most of the cases had long been mistaken for tuberculoid leprosy and it was their failure to respond to DDS that aroused the suspicion that this was a disease apart. The first few biopsy specimens we received showed a picture reminiscent of granulomatosis disciformis (Miescher), and Weber and I reported the condition under the name of Mkar disease. We were unaware that Leiker (1964) was also studying the condition and it was he who called it granuloma multiforme.

The lesions are usually multiple and are found mainly on the exposed skin of the upper half of the trunk and arms. Any dermatologist seeing the condition in a region where leprosy is rare would think granuloma annulare the likeliest diagnosis ; but a non-dermatologist in the leprosy belt could be excused for thinking of leprosy, usually tuberculoid.

The lesions of granuloma multiforme itch, a symptom most uncommon in leprosy and granuloma annulare ; there is no sensory loss nor any sign of leprosy in the histological picture which may be suggestive of granuloma annulare, granulomatosis disciformis, necrobiosis lipoidica or even sarcoidosis, and these are the diagnoses that would probably be suggested in patients found anywhere but in an area where leprosy is endemic.

Apart from the absence of nerve involvement a good histological point in differential diagnosis is that the granuloma of tuberculoid leprosy pushes the dermal fibres aside while that of granuloma multiforme causes degeneration of the dermal fibres in the infiltrated area (Marshall *et al.* 1967).

In the region of Nigeria surveyed by Leiker the incidence of leprosy is 1.5 per cent and of granuloma multiforme 1.7 per cent, but the two diseases are very seldom found in the same patient. At first it was thought that the disease was relatively confined to the Tiv area of Nigeria, but Leiker has since found many cases in East Africa and there may be other foci in Indonesia and Indochina.

The cause of the condition is unknown and treatment is ineffective. The points about it that take my fancy are that it seems to be prevalent only where leprosy is also prevalent, and that the two conditions are usually mutually exclusive. Somebody suggested that it might be caused by bats' dung falling on the patients, but this I consider far-fetched.

The only suggestion I can make is that it might be a manifestation of leprosy in people with a very high degree of resistance.

POSTINFLAMMATORY ELASTOLYSIS AND CUTIS LAXA

Over the past eight years we have seen six cases of an unusual disease in which chronic, recurrent inflammatory lesions of the skin eventually end in a state of cutis laxa (Marshall *et al.* 1966). The disease is not peculiar to Africa and odd cases have been described in Europe in the past. A month ago I saw a typical example of the disease in a white boy, 3 years old, in Rio de Janeiro.

All our cases were in Coloured children, 5 girls and a boy, from the vicinity of Cape Town and none older than 3 years when the disease began. None had ever been in contact with any of the others.

The cutaneous lesions were identical in all. The primary lesion is a bright red papule that quickly extends to form a plaque with a red advancing edge and a dark bluish-red centre. The centre subsides and becomes wrinkled and hyperpigmented. Confluence of plaques produces large lesions with circinate margins. Lesions at first may resemble those of erythema multiforme, later they are reminiscent of erythema chronicum migrans or erythema annulare centrifugum. The face, ears and neck are always affected at some time, but any part of the surface except the palms and soles may suffer. The disease remains active for months at least, sometimes years and healing leaves loose skin which may give the child an ancient appearance if the face is badly affected. The laxity of the skin is never as pronounced as it is in cutis laxa of the congenital type.

Two patients had pneumonia early in the disease, but none shows any sign of systemic elastolysis.

The histological picture is at first of oedema and a neutrophilic infiltrate densest in the deeper dermis and around blood vessels and appendages. There is degeneration of elastic fibres which becomes marked in the final phase of cutis laxa when there is also some degeneration of collagen.

No cause has been found and the effects of treatment with steroids, antibiotics and other less orthodox remedies are so poor that we now leave well alone.

LICHEN PLANUS

Lichen planus in white people in Africa usually follows the standard patterns seen in Europe, but the disease in people with darker pigmentation may be different.

Negro patients in the tropics often have lesions more widespread and more exuberant than those commonly seen in Europeans. Basset (1959) has described the lesions as resembling those of a European seen through a magnifying glass. Lesions of the buccal mucosa are very seldom seen in the Negro in any part of Africa, and are relatively rare in the Coloured (mixed) population of South Africa.

In Egypt, El-Zawahry (1969) has described a lichen planus with lesions confined largely to the exposed skin of the face, neck, hands and arms. He considers both light and heat to be precipitating factors and for this reason has called the condition lichen planus tropicus rather than actinicus which, to my mind, would have been more apt.

The same phenomenon was described earlier in Palestine by Dostrovsky and Sager (1949) who found it in 51 out of 131 patients with lichen planus seen over a period of 16 years. They thought that the condition was commoner in dark skinned people than in fair. This variant of lichen planus has also been described in the Argentine (Kaminsky *et al.* 1956). I see it in South Africa, usually in Bantu or Coloured patients, but it is not so common as in North Africa or the Middle East.

All over Africa pigmented lesions or their remains are, not unexpectedly, commoner than in Europe. Lichen planus-like eruptions due to medicaments such as Mepacrine and colour film developers have been seen in Africa in people of all shades of complexion. In one case with lesions largely confined to the hands and wrists we suspected that an antibiotic, Tylosin, handled by the patient might be implicated.

When I started collecting information about skin diseases in Africa I was told by correspondents from the tropics that lichen planus seemed to be getting more prevalent. I took no notice as I thought that the disease was simply being recognized for the first time. Recently, however, Privat and Faye (1967) have reported that 5 per cent of new patients seen at the hospital in Dakar, Senegal, in 1966 had lichen planus. The figures seem to have risen in Dakar even since 1961 when Basset (1964) found the incidence of all papulosquamous and lichenoid eruptions to be 4.9 per cent. In Lagos, Nigeria, Clarke (1962) reported the incidence of lichen planus at 1.1 per cent in 1950 and 1 per cent in 1960 which is about the average for hospital statistics in all continents.

In Senegal the disease is one of young city dwellers more than of country people. Privat and Faye speculate on the old theme of psychosomatic disease, but I am inclined to think of some more tangible thing, perhaps a photosensitizer, encountered in the new environment.

BURKITT'S TUMOUR

Burkitt's tumour, multifocal lymphoma, is a common cancer of children in tropical Africa and the jaws are a common site of election for lesions (Burkitt, 1968).

It was first thought to be confined to those parts of the wet tropics where the temperature never falls below 60°F. or the annual rainfall below 20 inches ; in other words the region suitable for the wet tropics group of mosquito vectors.

However, once Burkitt had described the condition others soon found it elsewhere in Africa and in other continents in a variety of climates. Hoogstraten (1967), for example, recently claimed that it is the commonest tumour of children in central Canada.

Nevertheless, it is a tumour that deserves all the interest it is receiving and it may well be caused by an infective agent. Cases develop early in life in people living in high incidence areas in Africa, but only later in people who migrate from low to high incidence areas.

Response to treatment with cytotoxic agents is often spectacular in early cases with lesions confined to the jaws and long remissions suggest that actual cure may be possible.

KAPOSI'S SARCOMA

Before the second world war Kaposi's sarcoma had been recognized only a very few times in black Africa and it was then described as a disease of Jews and Italians because most cases were found in people who lived, or had been born, in that region of Central Europe that stretches down from Lithuania and Latvia through Poland and Austria and northern Italy to Corsica. After the war it was recognized as one of the commonest cancers of Negroes in Africa and it seems, in fact, to be more prevalent there than it ever was in Europe (Marshall, 1964). The disease is not new in Africa ; it was always there unrecognized as Oettlé (1962) showed when he re-examined old histopathological specimens at the Institute for Medical Research in Johannesburg.

The distribution of the disease in Africa is interesting. The epicentre is in the Congo where 12 per cent of cancers are Kaposi's sarcoma ; that is according to histological diagnosis. The incidence declines as one leaves the epicentre and falls to between 1 and 2 per cent of cancers in South Africa and Senegal.

It is unlikely that an infective factor is involved as even in a multiracial land like South Africa the disease is predominantly one of Negroes and seldom affects their white or Coloured neighbours ; no Indian in South Africa has yet been affected.

The disease in Africa is much the same as it is in Europe except that Negroes seldom have other diseases of the reticuloendothelial system as well as Kaposi's sarcoma while such combinations are often reported in Caucasians. Negro children quite often have systemic Kaposi's disease with little or no skin involvement.

As to treatment, I would counsel that the chronic variety of the disease, especially in elderly people, be left in peace if it is causing no bother.

TREPONEMATOSES

My remarks about the treponematoses concern the academic more than the practical aspects. The general concept today is that the treponematoses are all caused by organisms that developed from a single ancestral treponeme by mutation and survival of species in various environments (Willcox, 1969). The treponemes pathogenic in men can still not be differentiated by any microscopic or serum test and even some nonpathogenic treponemes may cause the same immunological reactions as do the pathogens.

A discovery of great interest is that certain monkeys in tropical Africa harbour a treponeme, called after its discoverer *T. Fribourg-Blanc*, in their popliteal lymph nodes. In these monkeys it causes no symptoms or surface lesions, but it does excite immunological reactions. It is pathogenic when inoculated into the skin of other monkeys (macacus of Asian origin) and produces lesions akin to those of yaws rather than those of venereal syphilis. When one recalls that a treponeme, *T. zuelzueriae*, with antigenic properties, is found as a saprophyte in mud one may speculate about its relationship to the asymptomatic popliteal node affection of African monkeys.

There is little likelihood that the treponematoses will soon be conquered in Africa which is everywhere vastly under-doctored outside the cities. In South Africa the upswing of venereal syphilis has come a little later than it did in Europe, but it is now in full swing and is accompanied by a return of endemic syphilis in country districts.

VIRAL DISEASES

New viruses and viral diseases are constantly being discovered in tropical Africa. One newcomer with skin lesions is o'nyong nyong fever which is rather like dengue and produces a measly rash and symptoms of joint and muscle pains; it is quite benign and runs its course in a week.

In South Africa there have been a few outbreaks of epidemic follicular keratosis over the past 20 years. Findlay and Whiting (1968) have recently suggested that these may have been caused by Sindbis or West Nile viruses which are harboured by man and wild birds and spread by mosquitoes.

FUNGUS DISEASES

Mycetoma is nothing new, but we might note that it is most prevalent in a belt across Africa between Dakar and Djibouti where the climate favours the growth of the micro-organisms and of bushes with thorns to introduce them under the skin, oftenest on the naked foot (Rey *et al.* 1962). The condition is found in South Africa, but less often than it was 50 years ago, presumably because more people are now shod.

African histoplasmosis, which is a relatively benign disease that affects mainly skin and bones and seldom the lungs, now seems to be accepted as a disease apart. The African disease is caused by *H. duboisii* which is larger than *H. capsulatum*. Vanbreuseghem (1969) has covered this subject so fully that I need say no more.

Black piedra is worthy of mention because it is common in apes and monkeys in Africa but seemingly rare in man. Only one proven case has been found, oddly enough in a white child with Netherton's bamboo hair syndrome (Marshall and Brede, 1961).

METABOLIC DISEASES

Although odd cases have been found throughout Africa, the hepatic porphyrias are much more prevalent in South Africa than elsewhere. The whites usually have familial porphyria variegata, the Bantu acquired hepatic porphyria and the Coloured people may have either. What strikes me as odd is that the two varieties are so common in one area and I wonder if there is not some local environmental factor that may precipitate symptoms in people predisposed by either genetic or other influences (Marshall, 1963).

South Africa also has a relatively large number of white and Coloured people with lipoid proteinosis and recent genealogical studies suggest that the condition was brought to Africa by a German family in the 17th century (Heyl, 1968 ; Gordon *et al.* 1969).

CONCLUSION

In conclusion I would like to remark on some problems concerning skin colour. We still do not know enough about the anatomical and physiological differences between black and white skins and the intermediate shades, let alone about other genetic factors which may influence the distribution of diseases in different races.

The question of racial pigmentation has been re-examined of late and there are some new theories as to why the native inhabitants of the tropics are dark skinned. It is unlikely that dark skin was an adaptation simply to provide protection against some diseases, notably tumours, caused by exposure to the sun, but not liable to have led to the extermination of a race.

Loomis (1967) still thinks skin colour was an adaptation to climate but he relates the distribution of the races to solar ultraviolet radiation and vitamin D synthesis. In northern latitudes there is selection for white skin that allows maximum synthesis of vitamin D and protection against rickets ; in southern latitudes there is selection for dark skin that protects against overproduction of vitamin D and its toxic effects.

Wassermann (1969) thinks tropical disease rather than climate was the major factor in producing racial pigmentation. Increased activity of the reticuloendothelial system was necessary to cope with the bacterial, viral and parasitic diseases of the tropics. The activity of the reticuloendothelial system is inversely related to adrenocortical activity whose depression would lead to increased pigmentation. There is anatomical and physiological evidence of decreased adrenocortical activity in African and American Negroes and in Indonesians as compared to whites, and Wassermann suggests that a single enzyme deficiency, genetically controlled, may lead to lesser adrenocortical function and thus increased reticuloendothelial activity which may have survival value in the tropics.

It is often stated, and I think with reason, that the black skin is less liable to allergic contact dermatitis than is white, but no good explanation for this has been advanced. There is no proof that a thicker stratum corneum or melanin itself offer protection. Wassermann has an idea about this subject too, and it sounds quite attractive provided you accept that the basophil cells of blood and tissue are related or identical and that mast cells can manufacture melanin. The Bantu people in South Africa have a tendency to basopenia and frequently show basophilia in the inflammatory response in the skin. Macrophages in the inflammatory response contain either melanin or basophil granules, seldom both. If mast cells in the black skin are largely committed to producing melanin they would be less available to participate in allergic reactions.

As interesting at present as the discovery of new diseases is the changing spectrum of the dermatoses caused by the industrial revolution and the shift of population from the countryside to the cities. The effects of a little more hygiene and a little more education and better feeding in the more affluent city society are soon evident. Some changes, like the reduction of contagious diseases are for the good ; others such as exchanging endemic syphilis for venereal syphilis and the increasing incidence of diabetes are not so good. In general it may be said that African Negroes living in cities are tending to adopt the disease patterns of the whites.

Arising from what I have said about new diseases, is it not time in this computer age that we had a standard system of nomenclature and a central register fed with information, old and new, that could be consulted by anyone turning up something unusual?

I would remind you that I am still collecting information about African diseases for my own little register at Stellenbosch University and welcome new contributors.

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