AINHUM

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As the number of coloured immigrants into the United Kingdom increases we must expect to see more examples of the few diseases that are peculiar to negroes. Among these is ainhum. In some tropical areas this is by no means a rare disease, for there were over 200 cases recorded in medical literature before the second world war. So far, however, only one case has been reported in the British Isles. We are reporting a second, study of which has enabled us to throw a little new light upon the pathology of the condition.

This interesting disease is confined to the toes, to negroes and, almost exclusively, to the male sex. There are no reports of indisputable cases in females except that of Goehring (1944), and none at all in half-castes, in Indians, or in other dark-skinned peoples. The large majority of true cases of ainhum have occurred in patients aged between twenty and fifty years.

There is some doubt about the origin of the name. It is usually thought to be derived from the Nago or Yoruba word for “to saw” or “to cut” (Castellani and Chambers 1919).

The clinical picture is unmistakable. A constricting sulcus appears around the base of one fifth toe and later, usually, around that of the other. The sulcus deepens, the toe becomes progressively externally rotated, its connection to the foot becomes increasingly slender, and if it is not amputated it eventually drops off.

CASE REPORT

A West African negro, born in Lagos in or about 1911, was transferred to the Manchester Royal Infirmary from the Manchester and Salford Hospital for Skin Diseases in March 1956. He had lived in Lagos until 1944, when he became a merchant seaman and visited Britain for the first time. At no time had he gone barefoot and he had always lived in cities. In 1947 he settled in Manchester as a café proprietor, and he has not left England since.

In 1954 he noticed a shallow, linear groove in the skin of the dorsal surface of the base of his fifth right toe and, shortly afterwards, of his left fifth toe also. On the right side the groove became progressively deeper, on the left less so. After about two years the right toe gave him some pain.

He had had no important previous illnesses. He gave no family history of similar trouble. He had, in fact, never heard of ainhum.

The state of his feet on admission to hospital is shown in Figure 1. The sulcus around the right fifth toe appeared mildly inflamed. It encircled the toe at the level of the proximal end of the middle phalanx. Distal to the sulcus the toe was swollen and the skin of its plantar surface had a glazed appearance. The groove on the left toe was shallower and did not quite encircle the toe on the plantar surface. Both fifth toes were warm. Sensation in them was normal and, though voluntary movement was impossible on the right side, it was performed fully on the left.

There was a scaly patch on each side of his neck. These patches had been unchanged for twenty years, and were neither infiltrated nor anaesthetic. No other lesions could be found.

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The blood was normal. The Wassermann reaction was negative. Scrapings from the sulci and from between the other toes did not produce any fungus, neither did scrapings from the lesion of the skin of the neck. Culture of a smear taken from the inflamed sulcus on the right fifth toe grew a staphylococcus, sensitive to chloramphenicol and to erythromycin.

Radiographs (Fig. 2) showed that the sulcus on the right side was almost amputating the fifth toe in the region of the proximal interphalangeal joint. The appearances were those of erosion of the head of the proximal phalanx. The bony outlines were normal on the left side.
side. On both sides, the middle phalanges, which are the bones most commonly attacked by ainhum (Spinzig 1939), looked normal. These appearances are shown more clearly in the radiograph of the amputated toe (Fig. 3).

![Fig. 2](image1)
Radiographs of the feet.

![Fig. 3](image2)
Radiographs of the amputated right fifth toe.

The right fifth toe was amputated through the metatarso-phalangeal joint. The cut surfaces bled normally and healed well. The patient refused any form of treatment for the left fifth toe.
Histological sections of the whole toe were taken in a plane running longitudinally through the nail, and in a plane at right angles to this surface. The constriction lay at the level of the proximal end of the middle phalanx and was deeper on the volar aspect, where the sulcus contained much keratinous debris (Fig. 4). The epidermis covering it was intact at the three sites examined, though it was compressed at the apex of the sulcus. In the region of the constriction, the zone between the epidermis and the bone or the joint capsule consisted of two distinct layers. The more superficial of these layers was relatively free from inflammatory infiltration; the morphology and orientation of its fibre bundles were like those of normal dermal tissue. The deep subcutaneous layer, by contrast, was oedematous, deficient in fat and infiltrated with plasma cells and with lymphocytes. These cells were particularly related to areas of perivascular oedema. The fibre bundles in the deeper tissues, though disrupted here and there by cellular infiltration, were mainly orientated longitudinally (Fig. 5). Numerous gram-negative diplococci or cocco-bacilli were found in a small area of the granulation tissue deep in the volar subcutaneous tissues just proximal to the sulcus. The significance of this finding is obscure because organisms were not found in other parts apparently equally severely affected.

The inflammation in the deep tissues was greatest in the region of the proximal...
interphalangeal joint, but it extended from the metatarso-phalangeal joint distally as far as the terminal joint.

Both interphalangeal joints and, to a lesser extent, the metatarso-phalangeal joint, were involved in a chronic inflammatory process apparently continuous with that in the deep tissues. This resulted in an erosive arthropathy histologically indistinguishable from that seen in rheumatoid arthritis (Fig. 6). Inflammatory change also involved the periosteum; new periosteal bone had formed on both phalanges (Fig. 4).

**DISCUSSION**

Ainhum was first described in 1860 by Clarke, “late Surgeon to the Natives” of the Gold Coast. He called it “dry gangrene of the little toe of negroes.” It was first recognised as a distinct disease by da Silva Lima of Bahia in 1867, and his account of the clinical features has not yet been surpassed. The disease has been reported in all parts of West Africa, in the West Indies (Doyle 1889, Jiménez 1946), in Panama (Kean and Tucker 1946, Kean, Tucker and Miller 1946), in South America and at least fifty times in the United States of America, mostly in the Southern States (Spinzig 1939, McKnight 1940, Montgomery 1942, Spencer 1942, Cornbleet 1946, Hersh 1946, Omens 1946, Tye 1946, Goehring 1944, Hucherson 1950 and Stack 1950). Several of the reported cases have occurred in negroes born in the United States. Indeed Matas (1896) stated that it was “not prevalent” among the African slaves originally imported into that country.

The disease was seen in a Krooman serving in the Royal Navy in 1917 (Barton 1918) and in a fifty-five-year-old negro in Bristol in 1941 (Davies and Hewer 1941). We have not found any other report of a case in the United Kingdom.

The fifth toe has been affected in all reported cases, and in most instances both fifth toes. It is unusual for the disease to reach the same stage in each fifth toe at the same time. Involvement of the fourth toes may rarely follow the development of the disease in the fifth; in a very few cases (Kean, Tucker and Miller 1946) there has been involvement of the third, fourth and fifth toes of both feet.

A strong family history has been given by some patients (Duhring 1884, Simon 1921, Weinstein 1912).

Ainhum has been much confused with other diseases. Such confusion accounts for the reports of its occurrence in women and girls, in white persons, or in the hands (Wigley 1929, Drummond 1939, Johnson 1941, Neumann 1953, Priessl 1949, Sutton and Sutton 1935, Findlay 1951, Wells and Robinson 1952, Bluefarb 1948). There is no doubt that these reports in fact describe cases of congenital annular constriction of digits. Confusion with leprosy (Montestruc and Caubet 1947) is perhaps to be expected, for ainhum has been reported in persons suffering also from leprosy (Matas 1896). However, none of the forty-five cases considered by Kean *et al.* showed any evidence of leprosy, and none of the 110 negro lepers whom they studied post-mortem had ainhum. At least nine of the reported cases of ainhum had positive Wassermann reactions (Wright 1924, Kean *et al.* 1946), but the large majority of the others showed no evidence of syphilis. One case of alleged ainhum (Shaffer 1947) was almost certainly an example of diabetic gangrene.

The disease has occurred in sufferers from peripheral vascular disease (Davies and Hewer 1941) and in victims of syringomyelia (Tatz 1946). It has been regarded on rather scanty evidence as a variety of scleroderma (Acton 1928). It is often accompanied by hyperkeratosis of the plantar skin (Wigley 1929, Drummond 1939, Spencer 1942).

Ainhum is certainly not due to self-mutilation, nor to any form of injury. It occurs in negroes who have never gone bare-foot and in others who have never worn boots or shoes. It does not occur in non-negro people who dispense with footwear, even if they live in jungles. It is not due to infestation by parasites, as suggested by Wellman (1906).
THE NATURE OF THE DISEASE

Accounts of the histology of ainhum have been contradictory. Wright (1924) found evidence of endarteritis, but the Wassermann reaction was positive in his patient. Davies and Hewer (1941), who gave a full description of the histology in their case, also found thickening of the walls of the arteries and narrowing of their calibre, but their patient had generalised peripheral vascular disease. Ash and Spitz (1945) did not find any significant change in the vessels or in the nerves in a patient free from generalised arterial disease. Martens and Norris (1945) found evidence of osteomyelitis, and suggested that ainhum begins as such. Kean and Tucker (1946), on the other hand, found neither arterial disease nor osteomyelitis.

In our case, histological evidence indicates that the primary lesion is a chronic, non-specific and widespread concentric inflammation of the deep fascial layers and of the peritendinous structures of the toes. The erosive arthropathy and the periostitis can be explained as extensions of this process. The constricting band is apparently due to a purse-string effect of the deep inflammation on the normal dermal tissues. We cannot suggest how this effect is produced, or why the constriction usually appears in much the same site on every affected toe, though the site of the sulcus, which is not entirely free from variation, may merely indicate the site of origin of the inflammatory reaction. It certainly seems more likely that the clinical features depend on the curious anatomy of the inflammation rather than upon any unique etiological agent or unusual tissue response.

REFERENCES


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