NON VENEREAL TREPONEMATOSES YAWS, BEJEL AND PINTA Clinical Review and Management

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ABSTRACT

The authors summarise and illustrate the salient features of three non-venereal treponematoses, Yaws, Bejel (Endemic Syphilis) and Pinta, together with the current regimes for antibiotic treatment.

INTRODUCTION

The non-venereal treponematoses, Yaws, Bejel or Endemic Syphilis, and Pinta form a group of chronic bacterial inflammations constituting a major health problem in many third world countries. [1] Several cases of imported Yaws and Bejel have been reported recently in Europe.

Non-venereal treponematoses are transmitted by direct contact between children and adolescents living in poor social, economic and hygienic conditions. They differ significantly from venereal treponematoses in their mode of transmission, epidemiology and clinical presentation. [2] The clinical course progresses usually from primary lesions to secondary eruptions and then to late manifestations. Prenatal involvement and lesions of the cardiovascular and central nervous systems are not normally seen in non-venereal treponematosis infections but rare exceptions have been reported. [3]

Diagnosis of non-venereal treponematoses can be made on the basis of their typical clinical manifestations but it is desirable that they are confirmed by dark-field microscopical examination and appropriate serological tests such as Treponema pallidum haemagglutination antibody test (TPHA) and the fluorescent treponema antibody test (FTA-abs). [4] Recently a commercially available Captia syphilis-G immunoglobulin (GigG) Elisa test has been found to be a sensitive assay for treponemal antibodies in patients with Yaws.[5]

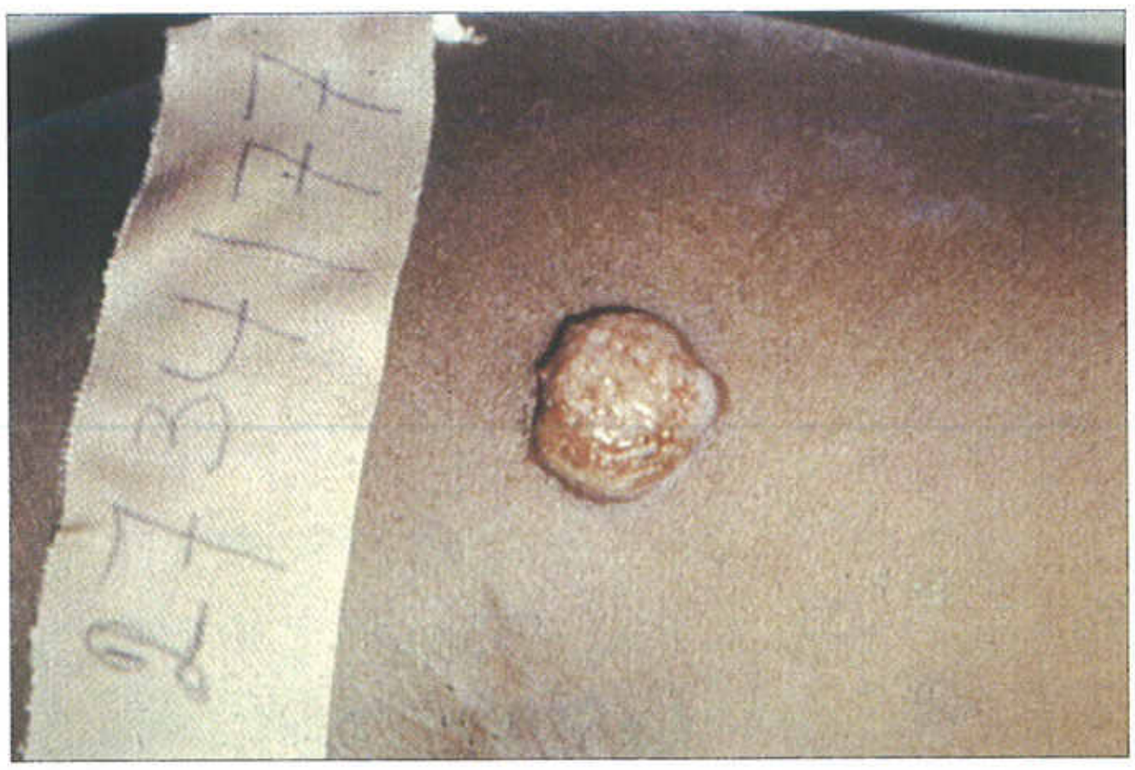
Also a Bio ELISa syphilis immunoassay has been found to be a sensitive and specific.[6]

YAWS

Yaws is an infectious non-venereal disease caused by treponema pallidum subspecies pertenue. It is predominantly a disease of childhood and poverty in hot humid climates with about 70% of infections occurring below the age of ten years. [7] It is not endemic in the Arabian Gulf region. [8]

Transmission occurs by direct non-venereal contact between a person with open primary or secondary lesions and a person with abraded skin. Most primary lesions occur on the feet, legs and buttocks. When genital lesions occur they are accidental rather than through sexual contact. [9]

The initial primary lesion ("Buba" or mother yaw) develops at the site of inoculation 9 - 90 days (average 21 days) from the time of infection. It begins as an erythematous infiltrated papule which ulcerates to form a yellow crusted lesion varying from one to five centimetres in diameter. [Fig.1] The lesion is neither indurated nor painful but is highly infectious. This stage may be accompanied by fever, joint pains and regional lymphadenopathy. After several weeks this mother yaw heals spontaneously leaving a hypopigmented pitted area surrounded by a dark halo.[10]



(Fig 1). Mother Yawî yellow crust ulcer

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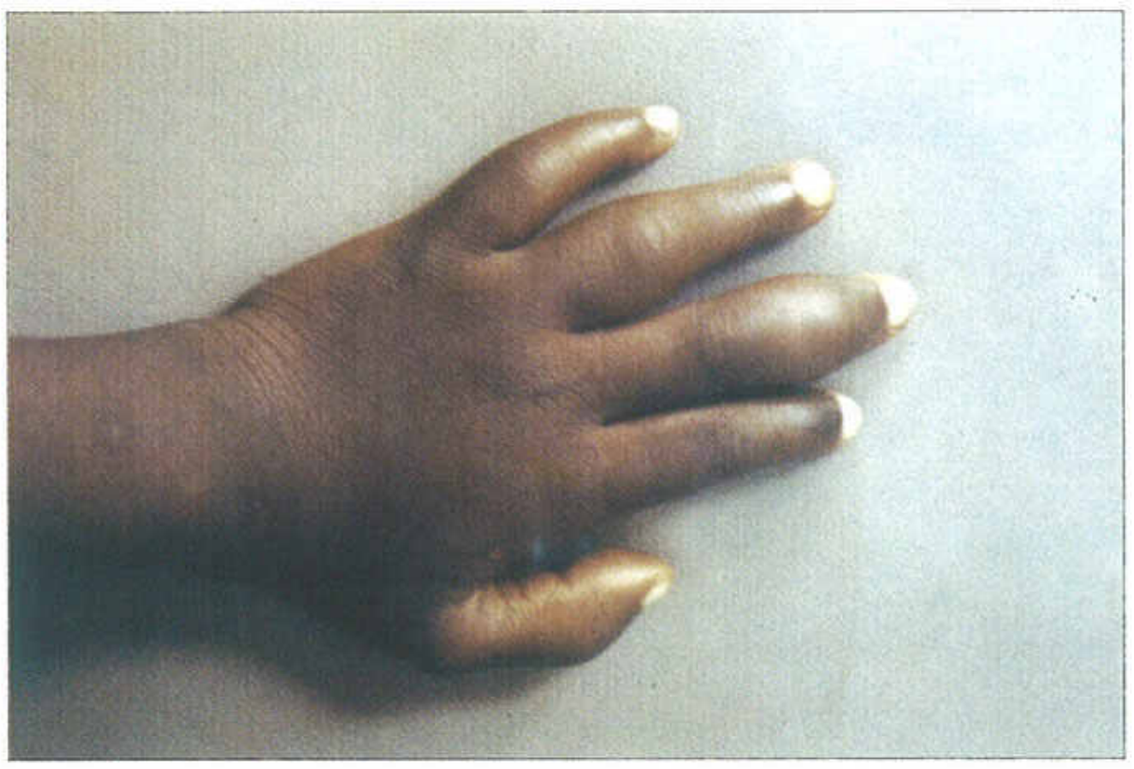
The secondary stage occurs six weeks to six months after the appearance of the mother yaw and is characterised by disseminated cutaneous lesions, palmo-plantar lesions, bone involvement and generalised lymphadenopathy. Constitutional symptoms of headache, fever, malaise and joint pain are common at this stage.[11] These secondary or "daughter" yaws teem with treponemes. They resemble the mother yaw but tend to be smaller and more widespread. [Fig.2] Macular and hyperkeratotic papules resembling secondary syphilis are found in the palms of the hands and on the soles of the feet.

(Fig 2) Daughter Yawî widespread lesions

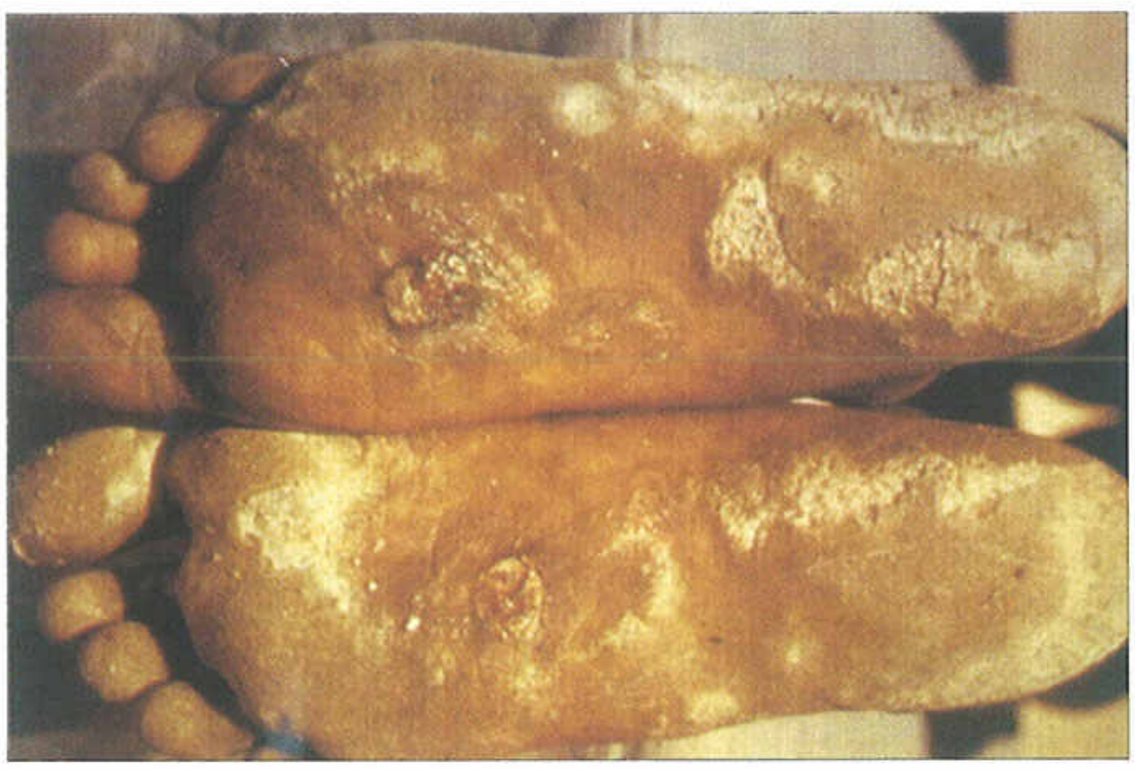
Condylomas lesions in the axilla and groin have been reported. Osteoperiostitis and polydactylitis cause a fusiform soft tissue swelling of the hands. [Fig.3]

Late yaws (tertiary stage) may occur several years after the primary infection in about ten per cent of cases. Keratoderma and hyperkeratosis of the palms and soles occur frequently .[Fig.4] Scarring and keloid formation following ulceration cause contractions and crippling deformities. [12] [Fig.5] Bone lesions at this stage include hypertrophic and gummatus periostitis and curvature of the long bones ("Sabre shins"). Bilateral swelling of the external aspects of the nasal processes of the maxilla (Goundou) is a characteristic lesion. [Fig.6]

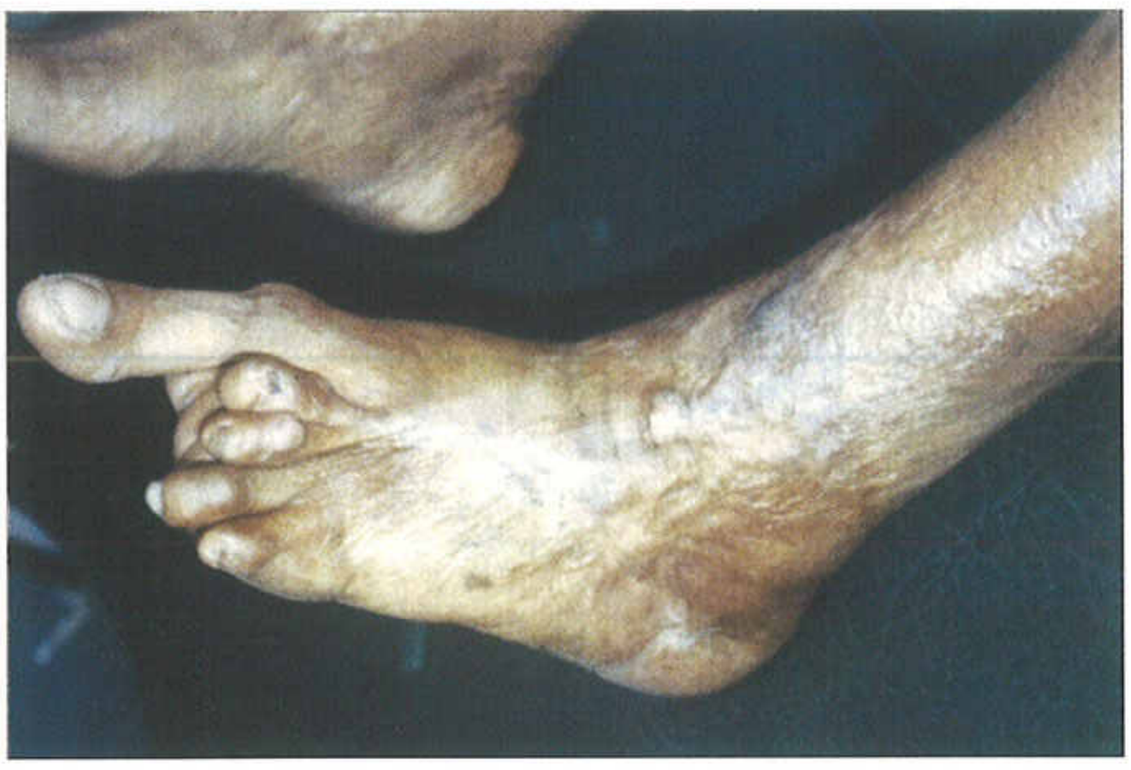
Gangosa also occurs in late yaws, appearing as a nasal perforation through



(Fig 3) Osteoporiostitis and polydactylitis of the hand.



(Fig 4) Keratoderma and hyperkeratosis of the soles



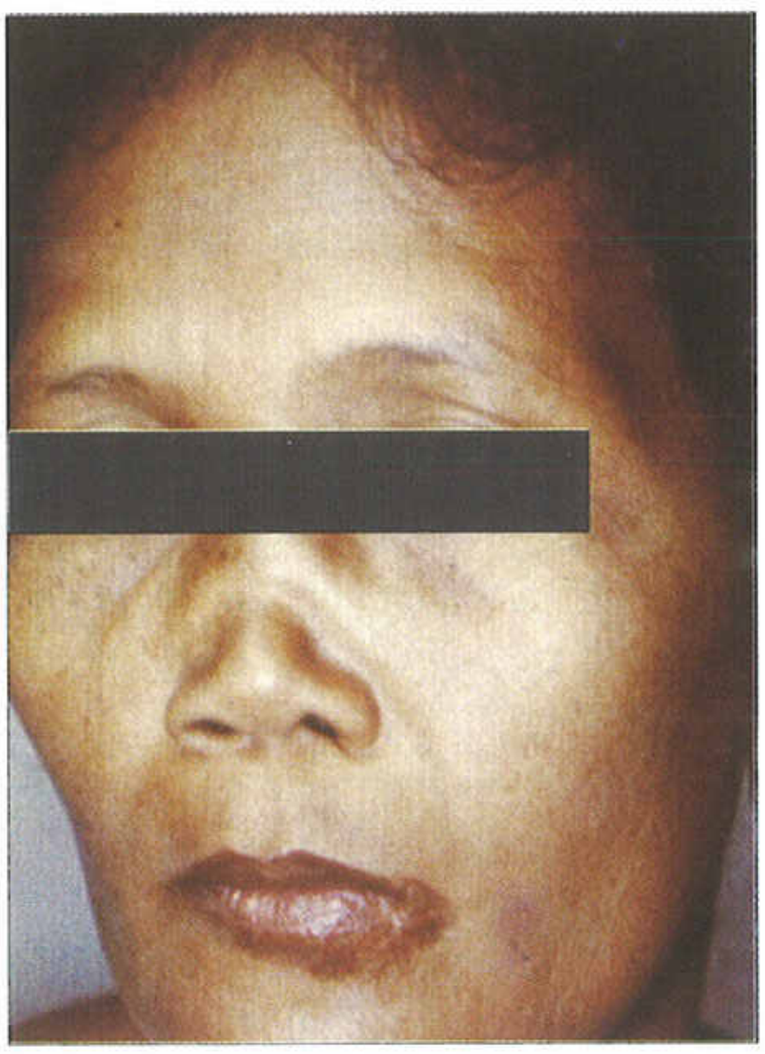
(Fig 5) Scars and keloids causing crippling deformity

direct extension of muco-cutaneous lesions at the border of nose and mouth. [Fig.7] Most reports suggest that the nervous system, including the eyes, is not affected by yaws. [13] The disease then enters a non-infectious latent period that may last life long.

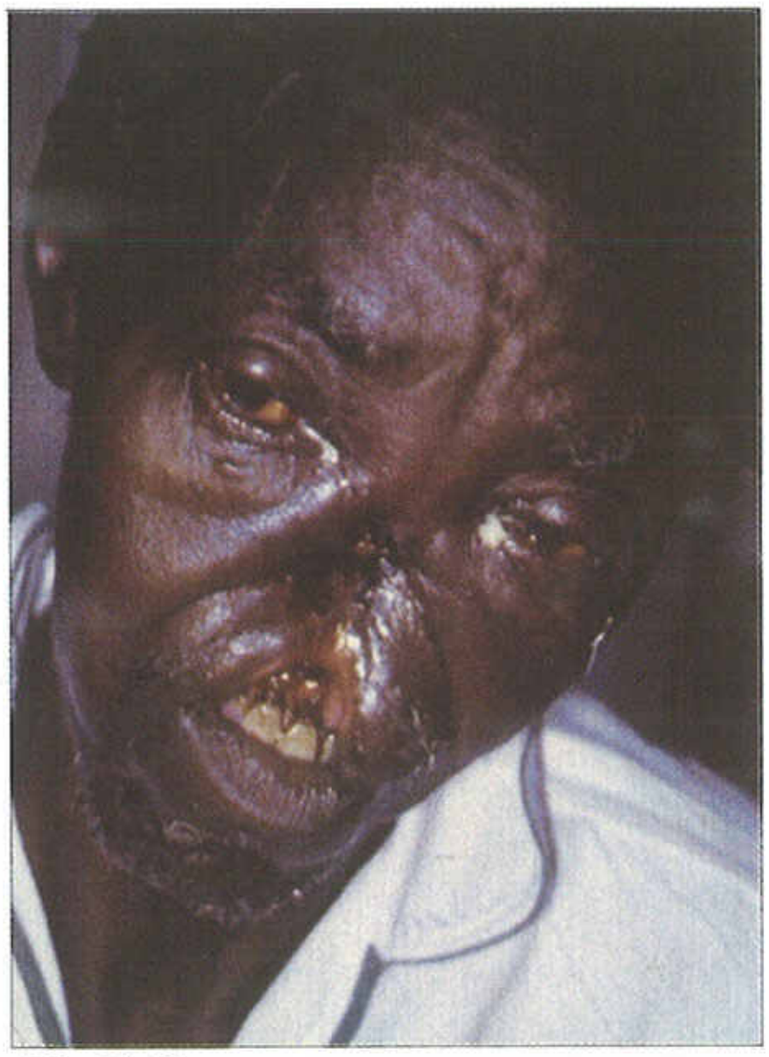
BEJEL (Endemic Syphilis)

Bejel is a chronic childhood infection of the skin, bone and cartilage, with the main reservoir in children under 15 years of age. The infection tends to run in families, being transmitted from child to child by close contact and kissing or by common contaminated drinking vessels passed from mouth to mouth. As a consequence most initial lesions occur on the oral mucosa. There is little evidence of congenital transmission.

Bejel has been found endemic in arid regions such as the Sultanate of Oman, the Kingdom of Saudi Arabia, Syria and Iraq. [14] In the United Arab Emirates venereal syphilis is uncommon but there is a high rate of seropositives in the 50-plus age group, thought to be due to old or attenuated Bejel. [15]



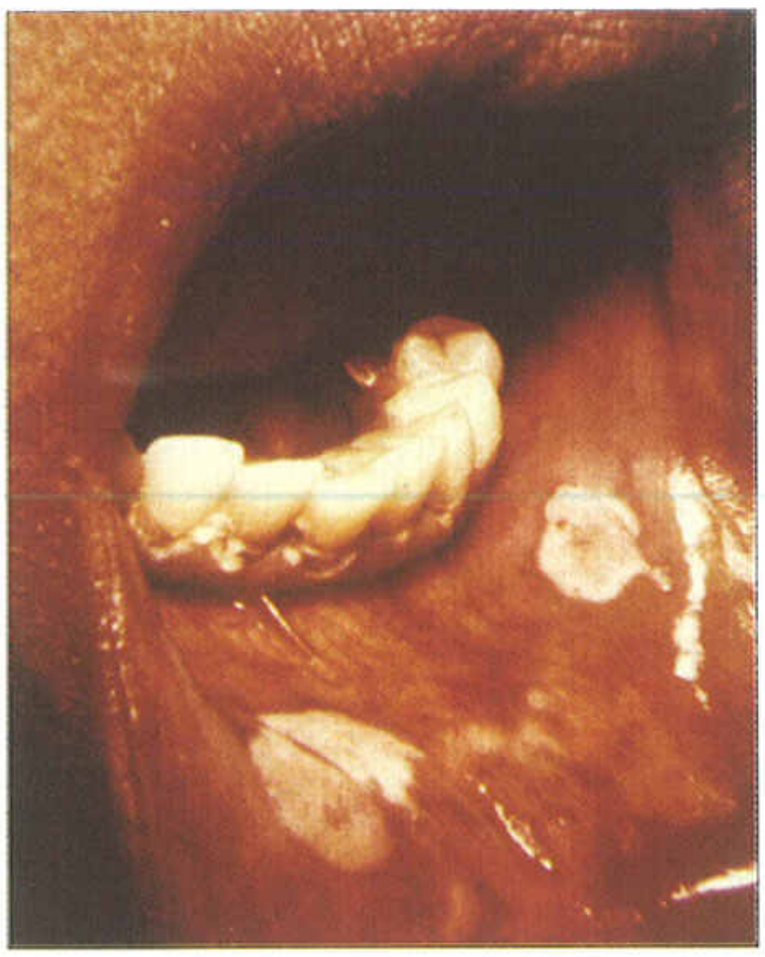
(Fig 6) Goundou.



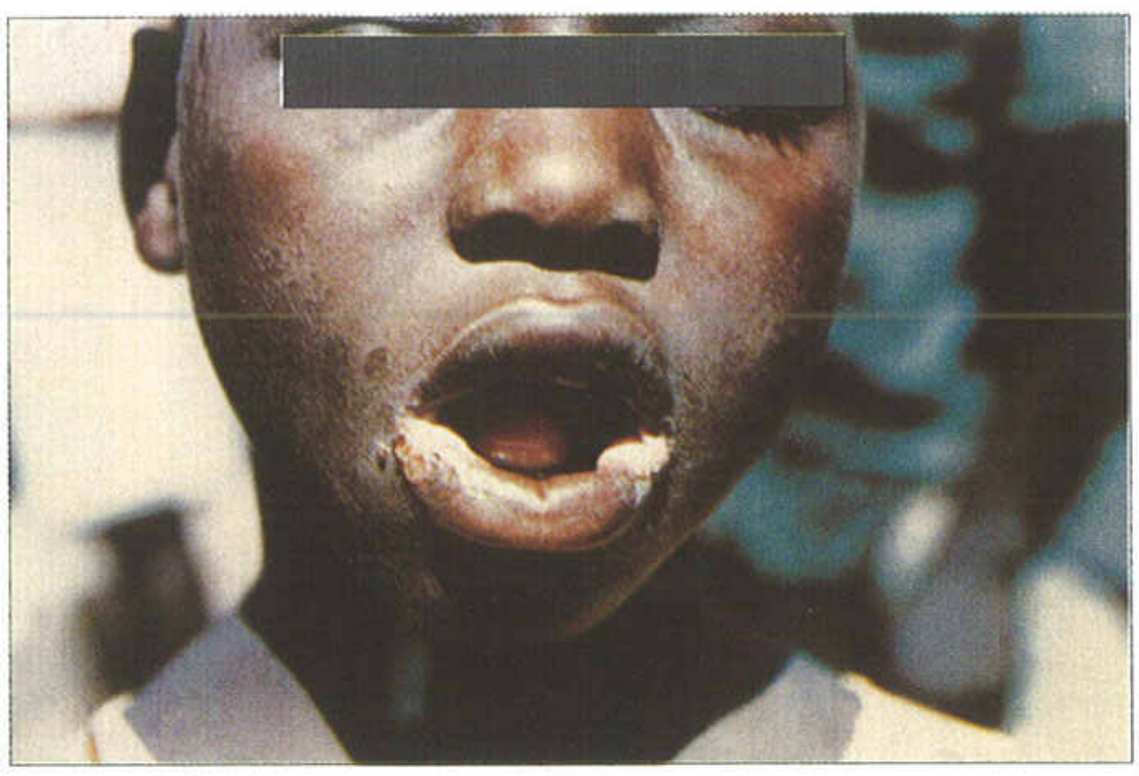
(Fig 7) Gangosa

The primary lesion of Bejel is a rarely noticed insignificant papule or ulcer on the oropharyngeal mucosa or on the nipple of the nursing mother. [16]

The most commonly seen secondary lesions ofBejel are mucous patches [Fig.8]; shallow painless ulcers on the lips, buccal mucosa, tongue and tonsils, frequently accompanied by a hoarse voice and regional lymphadenopathy. Angular stomatitis (Splits papules) occurs as a feature of the secondary stage [Fig.9] and non-pruritic disseminated papular, macular



(Fig 8) Mucous patches of Bejel



(Fig 9) Angular stomatitis (split papules)

eruptions, generalised lymphadenopathy, axillary and anogenital condyloma lata are other stigmata. Osteoperiostitis of the long bones causes nocturnal leg pain. Untreated secondary lesions persist for approximately seven to ten months. [12]

Tertiary signs of Bejel develop from six months to several years after inoculation. Gummata of the nasopharynx, skin and bones are common and destructive lesions of the palate and nasal septum are disfiguring features (gangosa-like) of the untreated disease. [17] Uveitis, choroiditis and optic atrophy occur frequently but there are no reports of neurological or cardiac involvement. [18]

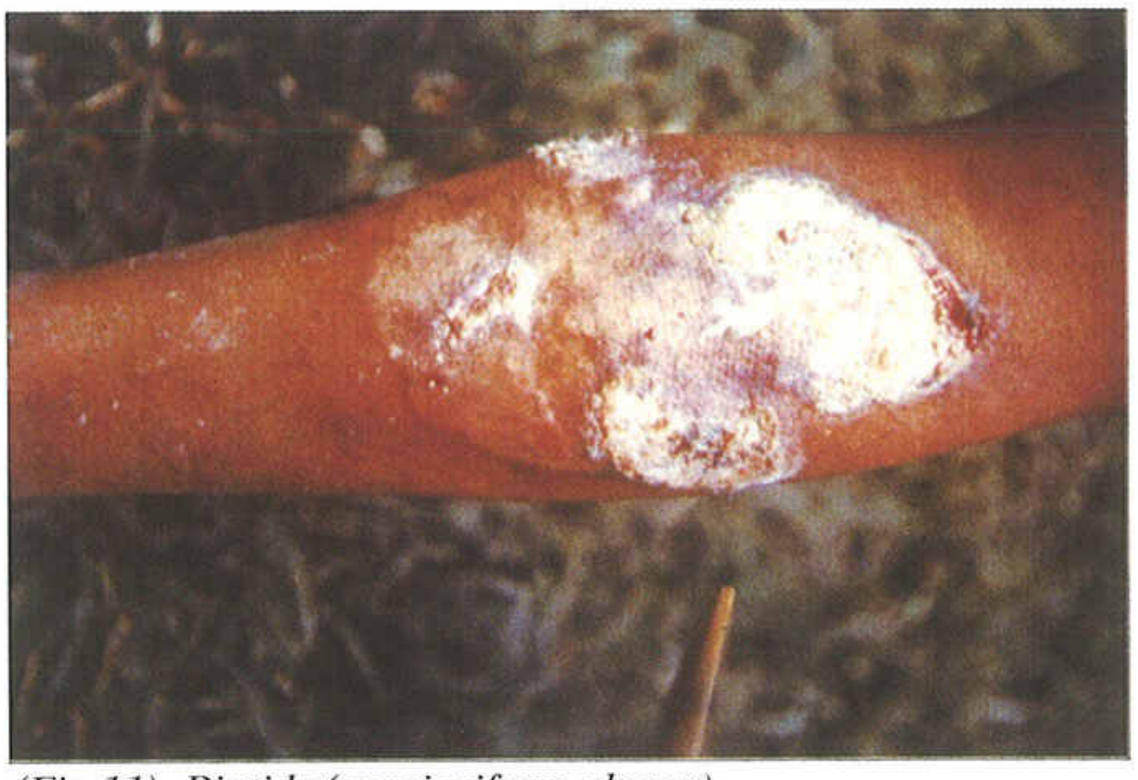
PINTA

Pinta, meaning a spot or mark, is unique among the spirochetal diseases in having skin manifestations only and in affecting persons of all ages. It is caused by Treponema carateum and 25 to 60 per cent of cases occur in children less than fifteen years old. [19] Like Bejel, Pinta is transmitted mostly between family members and "lesion to skin contact" is the usual method of transmission.

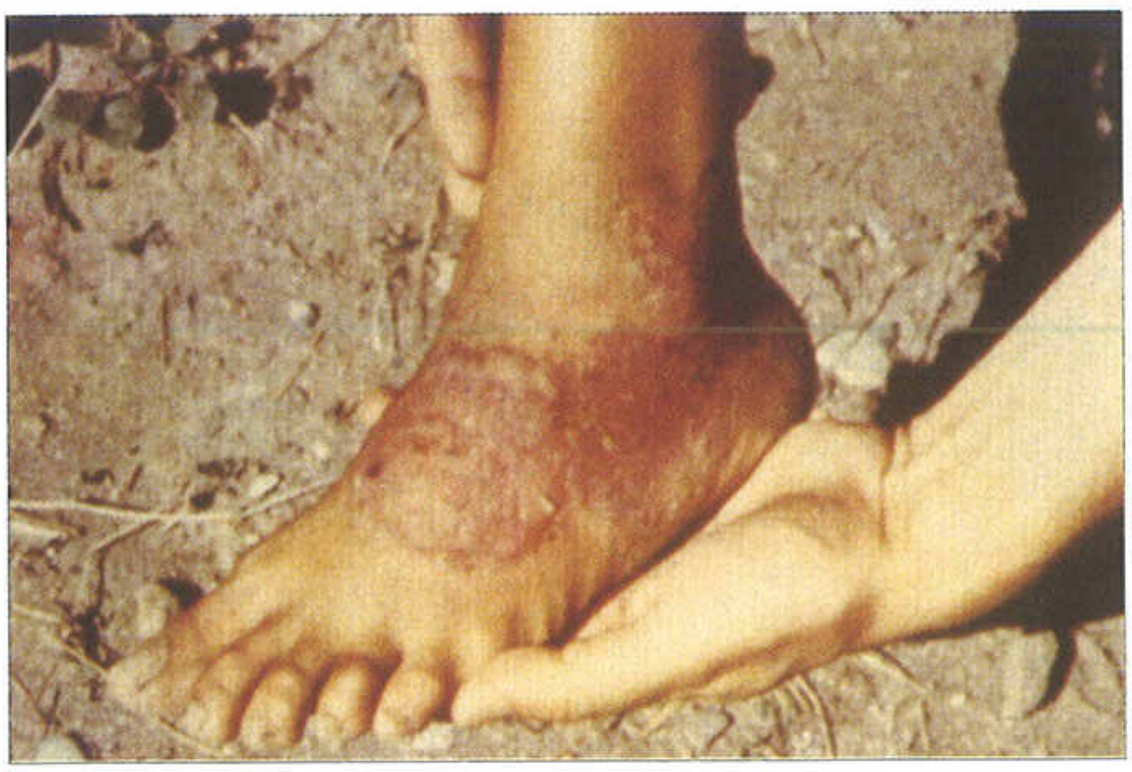
The initial lesion appears at the site of infection as a minute papule or as a macule surrounded by an erythematous halo six to eight weeks after inoculation. It may become an infiltrated plaque 10-12 cms in diameter, commonly on the lower extremities. [Fig.10]

The secondary lesions of Pinta ("Pintids") develop from months to years after the appearance of the primary lesion. They begin as small scaly papules that enlarge gradually and coalesce to form psoriasiform plaques in which several colours may be seen. [Fig.11]

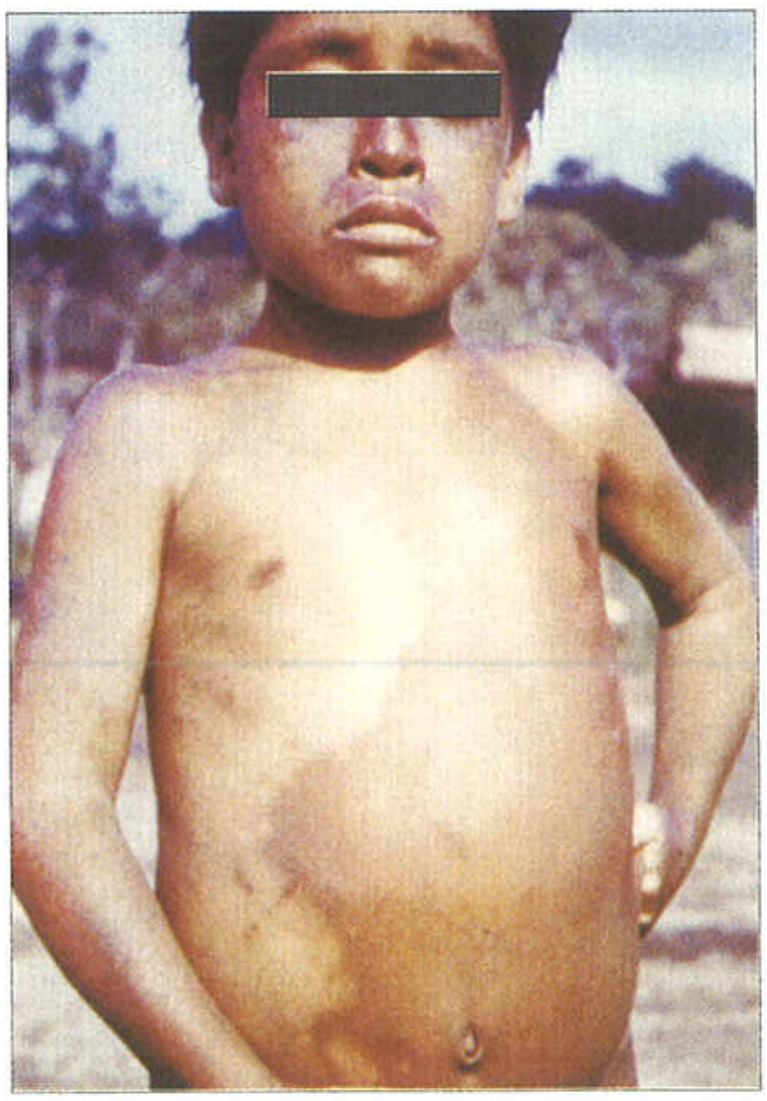
The late stage of Pinta is characterised by the development of depigmented lesions over the bony prominences, wrists, elbows and ankles. Symmetric achromic lesions tend to develop from three months to ten years after the appearance of the pintids. [Fig.12] Cutaneous atrophy and hyperkeratosis may also be present. There is no evidence of involvement of other organs. [20]



(Fig 11) Pintids (sporiasiform plaque)



(Fig 10) The initial lesion of Pinta



(Fig 12) Late pigmented Pinta

Treatment of Non-Venereal Treponematoses

Tissue injury occurring in the early stages of the diseases resolves completely with adequate treatment while tissue damage from the later stages is irreversible. For this reason treatment should be started as early as possible. [1]

Before antibiotics, non-venereal treponematoses were treated with preparations of bismuth and arsenicals. Today, 1.2 million iu of benzathin penicillin as a single dose is effective in adults and 0.6 million iu is effective in children less than ten years of age although the World Health Organisation recommends a single dose of 2.4 million iu in the early stages and three doses at weekly intervals in later stages of the diseases. Patients allergic to the penicillins can be given tetracycline at the rate of two grammes daily for ten days. The recommended alternative for children is oral erythromycin 10mg/kg four times daily for fifteen days. [21]

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