# A facial lesion ... the face of cutaneous tuberculosis

# Okello Armgod Blacka MB.Ch.B, MUST

## Introduction

Lesions of the facial skin can be difficult to diagnose in the absence of laboratory facilities. The patient described below is such an example where the clinician initially diagnosed facial kala-azar. It was only after the lack of therapeutic benefit and photographs were shared with international colleagues that the true diagnosis of cutaneous tuberculosis (TB) became apparent.

# Case presentation

# History

A 9-year-old non-immunized boy presented with a two years' history of an ulcer on the face. The ulcer progressed slowly beginning from the philtrum and enlarging peripherally to involve the nasolabial triangle as well as the cheeks (Figure 1). He had no history of cough, fever, night sweats or loss of weight. No other family members had similar lesions. He had visited the hospital on more than four occasions without any healing.

lymphadenopathy. Systematic examination revealed no abnormalities.

Based on the local experience of endemicity he was initially clinically diagnosed with muco-cutaneous kalaazar. Fluconazole and topical sulfadiazine was started.

Three months later, the lesion had advanced widely over the nasolabial areas and inter-orbital space with complete ulcerative destruction of the nasal bridge. Nasal orifices were partially obliterated. Ulceration had involved the left cheek, with the superior margin nearing the inferior eye-lid of the left eye. The inferior margins had involved the vermilion border of the upper lip with secondary exposure keratitis of the upper gum. He had difficulty feeding due to ulceration of upper lip. The rest of his body had widespread itchy papular lesions.

# Investigations

• Rapid Plasma Reagin (RPR) test for syphilis and Human Immunodeficiency Virus (HIV) serology were negative.





Figure 1a and 1b. The facial lesions on the patient

## Clinical examination

He was well nourished and did not look sick but covered his face with a towel. A BCG scar was absent. Face exposure revealed a regularly bordered, dirty excoriated ulcerative round lesion extending outwards. There were central crusts, plaques and fissures. It had pustular edges, not everted, undermined or fungating. There was no surrounding cellulitis. There was no regional

- Aldehyde gel test: positive. It should be noted that a positive test may also occur in tuberculosis, leprosy, syphilis and chronic malaria as well as in kalaazar.
  - ESR: elevated 86mm/hour

There were no facilities for a Mantoux test, Ziehl-Neelsen staining for Acid-Alcohol Fast Bacilli (AAFB) on skin scrapings or biopsy for histology.

## Diagnosis

Through the medium of this journal the photographs of this child's facial lesions were shared with a number

a Boma Hospital, South Sudan dr.okello@gmail.com



Figure 2. Two weeksafter the start of anti-tuberculosis treatment

of colleagues including Emeritus Professor of Tropical Medicine at the University of Oxford, Professor David Warrell. Cutaneous TB was considered to be highly likely and appropriate treatment was advised.

#### **Treatment**

The Standard WHO regimen was started with rifampicin, isoniazid, pyrazinamide and ethambutol for a two months' intensive phase (2RHZE). This will be continued for a further four months of rifampicin and isoniazid (4RH). This will be given as Directly-Observed Treatment (DOT)

# Outcome

The lesions started to heal rapidly within ten days of starting therapy (Figure 2). By three weeks there was almost complete disappearance of the skin ulcers with only residual mucosal lesions. At the beginning of the second month of DOT, the nasal orifice patency was improving allowing some nasal breathing. However he still breathes mainly by the mouth and has a hypo-nasal voice. He is currently in the second month of therapy (Figure 3).

#### **Discussion**

Cutaneous TB constitutes 1-2% of all extra-pulmonary TB cases in childhood and is often seen in association with malnutrition, low socioeconomic status and crowded living conditions. Cutaneous TB may develop due to *Mycobacterium tuberculosis* and *Mycobacterium bovis* and Bacille Calmette-Guérin [1]. There are a number of types of lesions:

- 1. Lupus vulgaris is the commonest. It tends to have sharply defined brown edges with a jelly-like consistency (apple-jelly nodules) and typically appears on the face around the nose, eyelids, lips, cheeks, ears and neck. It may follow inadequately treated earlier TB [2].
- 2. TB verrucosa cutis results from direct inoculation in a person who has had previous TB infection. The lesions appear brown-red and wart like especially on the



Figure 3. Six weeks after the start of anti-tuberculosis treatment.

hands, elbows, buttocks, knees and feet.

- 3. Scrofuloderma arises from TB infection of underlying tissues e.g. lymphnodes. These are painless and ulcerate.
- 4. Tuberculid occurs in patients with a degree of immunity to TB. It may appear as a nodular vasculitis on the back of the legs (Bazin's disease). Crops of crusting lesions also occur in a similar distribution to that found with TB verrucosa cutis.
- 5. Miliary TB may manifest with small red spots that ulcerate and form abscesses and is more commonly found in immunocompromised patients.

Two diagnostic criteria are used [3,4]:

Absolute criteria: Positive culture of *M. tuberculosis* but the rate of positive culture is low [5].

Relative Criteria are used in absence of positive cultures:

- Evidence or history of active tuberculosis at other sites.
- Clinical history and physical appearance.
- The presence of acid-fast bacilli.
- Tuberculous granulomas seen on histology.
- Positive Mantoux test.
- Response to anti-tuberculosis therapy.

Diagnosis of cutaneous TB is difficult because of these various clinical forms, the relative infrequency of this condition and hence the inexperience of the non-specialist. Cutaneous TB must be considered in all cases with chronic skin lesions because TB prevalence is high in South Sudan. Early diagnosis and treatment is extremely important in order to prevent complications. The rapid response to treatment is well demonstrated by this case.

Complications include metaplastic change (Marjolin's ulcer) into squamous cell carcinoma and basal cell carcinoma. Deformities such as destruction of the nasal

# **SHORT ITEMS**

bridge, palpebral destruction with secondary ectropion and exposure keratitis of eyes may also occur.

Where diagnostic facilities are inadequate as in South Sudanitis essential to take advantage of resources wherever they may be obtained. In this instance it was through this journal, the South Sudan Medical Journal, through which medium the clinicians were able to ask advice of tropical clinicians with experience in this field. The results of such international co-operation and friendship are clear to see (Figure 4).

# **Learning Points**

- The compilation of a differential diagnostic list is essential when considering such unusual presentations. Tuberculosis must always be included.
- We need to consider an unusual presentation of a common condition before thinking of a common presentation of a rare one. In this case, we first considered mucocutaneouskalaazar (typical lesion presentation, rare disease). In fact, TB skin (rare presentation, common condition) did not feature initially among the differential diagnoses.
- Seek advice and co-operation. This is now much facilitated by the e-communications now available leading to a swift response.

All photographs taken by the author and published with permission of the patient and his next of kin.

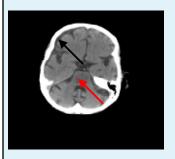
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# Case Study – Central Pontine Myelinolysis

## **Clinical History:**

Decompensated alcoholic liver disease and hyponatraemia. Relatively sudden onset of complex neurological symptoms including weakness, dysphagia, diplopia, disturbed consciousness and gait changes.



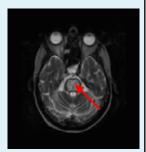


Figure 1

Figure 2

**Picture 1:** Non enhanced CT of the brain. This picture shows a hypodense appearance of the cerebral pons (red arrow) and a small right frontotemporal chronic subdural haematoma (black arrow).

**Picture 2:** Low resolution T2W image. This picture shows the extensive demyelination of longitudinally descending tracts and transversly crossing fibers in the basis pontis (red arrow).

# Radiological Report:

## CT Brain:

A non enhanced CT of the brain has been aquired. Picture 1 shows a hypodense appearance of the cerebral pons and a small right frontotemporal chronic subdural haematoma. The imaged portion of the fourth ventricle is unremarkable. No obvious space occupying lesions are seen within the cerebellopontine angle area bilaterally.

# MRI Brain:

A fast low resolution T2W sequence of the brain has been acquired due to restricted patient compliance. Picture 2 shows acute diffuse oedematous changes affecting the cerebral pons in keeping with a central pontine myelinolysis. There is no evidence of midline shift or of signs of raised intracranial pressure. The cerebellopontine angle area including fourth ventricle is otherwise unremarkable. Incidental note of a small fluid collection within the sphenoid sinus is made.

Contributed by Dr med. Stephan Voigt, Consultant Radiologist, St. Mary's Hospital, Isle of Wight, UK. <u>stephan.voigt@iow.nhs.uk</u>