## Brownie-nose: Hyperpigmentation in Neonatal Chikungunya

A neonate born to a mother with fever for a week prior to delivery was admitted with respiratory distress, and was treated with intravenous antibiotics for suspected sepsis. He developed marked hyperpigmentation over the face (*Fig.* 1) associated with worsening thrombocytopenia during the first week of life. This classical hyperpigmentation (brownie-nose appearance) suggested the possibility of neonatal chikungunya, which was confirmed by positive IgM antibodies to chikungunya in both the mother and baby. The platelet count gradually normalized and baby was discharged on day 10 of life.

Vertical transmission of Chikungunya, though rare, is well described with the maximum risk if the mother is viremic at delivery. Neonatal chikungunya is manifested by neurological, dermatological, ocular, renal and hematological involvement. Maculopapular and vesicolobullous rash, and striking hyperpigmentation are the common dermatological manifestations. Differential diagnoses include congenital lupus, drug rash (eg. imipenem), and bacterial (Listeria, *S. epidermidis*), fungal (Candida) and viral (human herpes virus 6, enterovirus) infections.



Fig. 1 Brownie nose appearance in neonatal chikungunya.

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## Kerion

A 5-year-old otherwise healthy boy presented to us with lesions over scalp, for 1 month. He had been treated with various systemic antibiotics and topical corticosteroids without any response. Cutaneous examination revealed a large mildly tender erythematous boggy swelling, associated with overlying crust (*Fig.* 1) and loss of hair over occipital area, extending towards the temporal region and post-auricular area of right side. There was sero-purulent discharge from few lesions. Nails and mucosae were normal. A KOH preparation made from the lesion showed fungal hyphae and the culture for bacterial growth showed growth of *Staphylococcus*. A diagnosis of Kerion was made and the patient was given prednisolon 1mg/kg/d



FIG. 1 Large erythematous boggy swelling associated with overlying crust (1a) and the healed lesion after treatment (1b).

for 2 weeks and griseofulvin 375 mg daily at bedtime for 6 week; the lesions showed marked improvement with regrowth of hairs in a few areas (*Fig.* 1b).

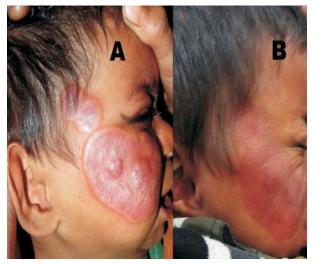
A Kerion is an inflammatory variant of tinea capitis as a result of overzealous host response leading to boggy, purulent plaques with abscess formation and alopecia. It is most commonly caused by *Trichophyton mentagrophytes* and *Trichophyton verrucosum*. Clinical differentials include pyodermas (lesions are painful, absent hyphae), folliculitis decalvans (pustular folliculitis leading to

scarring alopecia; fungal hyphae negative), langerhans cell histiocytosis (involvement of other organs system with a grave course) and dissecting cellulites of scalp (begins with deep inflammatory nodules, primarily over the occiput, that progress to coalescing regions of boggy scalp; fungal hyphae negative).

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## **Acute Febrile Neutrophilic Dermatosis (Sweet Syndrome)**

An 8-month-old boy presented with high grade fever of 7 days duration and few reddish lesions on face, scalp and upper limbs (*Fig. 1*). To start with, there were few lesions on the face that gradually increased in size and number to attain the present status. Drug history, past history and family history were non-contributory except for common cold and mild fever about 10 day prior to onset of cutaneous lesions. Cutaneous examination revealed multiple well-circumscribed erythematous to violaceous papulo-nodules and plaques (few targetoid) over the face, scalp and both upper extremities. Hairs, nails, and mucosa were unaffected; conjunctivae were congested. Laboratory



**FIG. 1** Well defined erythematosus to violaceous plaques and nodules on face (A) and flattening of lesions (B) after one week of treatment.

investigations revealed leukocytosis with neutrophilia and raised erythrocyte sedimentation rate. Punch biopsy followed by histopathological examination of a representative lesion from face showed dense perivascular neutrophilic infiltrate along with vasodilatation, nuclear dust and upper dermal edema, without any evidence of vasculitis. A diagnosis of acute febrile neutrophilic dermatosis was made, and prednisolone (1mg/kg/d suspension) was started under antiobiotic cover. Lesions showed improvement at the end of 1st week of treatment.

Acute febrile neutrophilic dermatosis, also termed Sweet syndrome (SS), is a reactive process characterized by the abrupt onset of tender, red-to-purple papules, and nodules that coalesce to form plaques. The plaques usually occur on the upper extremities, face, or neck and are typically accompanied by fever and peripheral neutrophilia. SS may be idiopathic or may be associated with infections, inflammatory conditions, malignancy pregnancy, or drugs. Clinical differentials include erythema nodosum leprosum in leprosy patients (presence of features suggestive of leprosy); cellulitis or erysipelas (mostly single lesion, boil prior to the onset of lesion); erythema multiforme (preceding drug history or infection, presence of target lesions); urticaria (transient, itchy) and leukemia cutis. The criteria for diagnosis have been proposed by Su and Liu, and revised by Driesch.

The treatment options include systemic steroids saturated solution of potassium iodide, dapsone and clofazimine, besides treatment of the underlying condition.

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