



Fig.1 Whitish crusting and fissuring in the perioral and perinasal areas with flaccid blisters in neck folds.

upper epidermis and presence of acantholytic cells in the subcorneal layer. A diagnosis of staphylococcal scalded skin syndrome (SSSS) was made. Patient had a complete recovery with peeling within 10 days on treatment with antistaphylococcal antibiotics.

SSSS, caused by *Staphylococcus aureus* exfoliative toxins (ET) A and B, generally affects neonates, infants,

and children less than 5 years of age, due to lack of protective antitoxin antibodies and immature renal function. Left untreated, large sheets of epidermis slough off to leave extensive areas of raw denuded skin that is sensitive and painful. The toxin is usually produced at a site distant from the lesions. ET acts as an atypical glutamate-specific serine protease that binds and cleaves desmoglein-1 (found in the upper epidermis, absent in the mucosa) which explains the specific site of action in the superficial epidermis and the absence of mucous membranes affection in SSSS. Cultures from the skin lesions are negative for staphylococcus in almost all cases. It is important to send swabs from other areas such as the umbilicus, nasopharynx and conjunctivae. Anti-staphylococcal antibiotics, temperature regulation, maintaining fluid and electrolyte balance, nutritional management and skin care form the basis of treatment. The main differential diagnosis remains drug-induced toxic epidermal necrolysis (TEN) the differentiating factors in TEN being adult onset, spared areas of the skin, mucosal involvement, presence of nikolsky sign only in involved skin (and not diffusely) and absence of perioral/perinasal crusting.

It is important to recognize this often dramatic looking skin disorder early, especially in nurseries, with the help of the above-mentioned classical features.

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Rowell Syndrome

A 13-year-old female child presented with multiple erythematous papules and plaques with some of them showing targetoid appearance (**Fig.1a**), along with a single large 8 cm x 10 cm sized polycyclic, erythematous plaque, involving right side of face, upper neck and external ear area (**Fig.1b**). Oral mucosa showed erythematous plaque involving right side hard palate with overlying multiple superficial erosions (**Fig.1c**). She had history of low-grade fever, photosensitivity, malar rash, Raynaud's phenomenon, chilblains and recurrent oral ulceration since 6 months. There was no history suggestive of recent infection or drug intake prior to onset of lesions.

Laboratory studies showed positive antinuclear

antibodies (ANA) in a speckled pattern at 1:160 dilutions, ESR 25 mm/h, and positive rheumatoid



Fig.1 Rowell syndrome

factor. dsDNA antibody, AntiRo and antiLa antibodies were negative. Hemogram, C3, C4, urine routine, urinary 24-hr protein, and renal function test were all within normal limits. Electrocardiogram and chest X-ray were normal. Histopathology of skin lesion was consistent with the diagnosis of systemic lupus erythematosus (SLE). Patient was advised photo protection and started on tapering oral corticosteroid along with Hydroxychloroquine. Lesions resolved after one month of treatment leaving only mild pigmentary changes with no relapse at 6-month follow up.

Rowell Syndrome (RS) is a unique clinical association of lupus erythematosus (LE) with erythema multiforme (EM) like lesions and a characteristic immunologic pattern. The etiopathogenesis is unknown. Diagnostic criteria include three major criteria: (1) LE

(systemic, discoid, or subacute); (2) EM-like lesions with or without mucosal involvement; and (3) speckled ANA; and three minor criteria: chilblains, antiRo or antiLa antibodies, and positive rheumatoid factor. For diagnosis all three major and at least one minor criterion should be fulfilled. Children with LE may present with EM-like lesions which must be differentiated from Classical EM which is often precipitated by factors such as infective agents or drugs and is not associated with any specific serological abnormalities or with chilblains. Treatment is with topical and oral steroids, dapsone, and antimalarial agents but response is variable with frequent recurrences.

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Pyogenic Granuloma

A four-year-old girl presented with an asymptomatic lesion over the right cheek of two months duration. History of precedent trauma and bleeding on manipulation was present. There was no drug intake or systemic complaints. Cutaneous examination showed a solitary, soft, non-tender, vascular papule with surrounding hemorrhagic crust over right cheek (**Fig. 1**). Systemic examination was normal. A clinical diagnosis of pyogenic granuloma was confirmed by excisional biopsy.

The term pyogenic granuloma (PG) is a misnomer, as it is neither infectious nor granulomatous. It is a reactive vascular nodule usually occurring after injury or irritation. PG is commoner in children and young adults, with no sexual predilection. PG may be associated with drugs like systemic retinoids, indinavir, etc.

Clinically, PG presents as a solitary, painless, red, brownish-red or bluish black papule/nodule. It is partially compressible without pulsation. Common sites affected are fingers, feet, lips, head, upper trunk, oral mucosa and perianal area. Spontaneous disappearance is rare. Histologically, a well circumscribed proliferation of small capillaries in a lobular pattern is seen. Differential diagnoses are hemangiomas, glomus tumors, warts and molluscum contagiosum. Hemangiomas are scarlet-red, non-tender, compressible, dome shaped papules/nodules present at birth/early infancy characterised by early rapid proliferation and spontaneous involution by 1-9 years of age. Glomus tumors are multiple, congenital, red/blue, less



FIG. 1 Pyogenic granuloma over cheek.

compressible, painful/painless papules coalescing to form plaques over any part of the body. Warts are multiple, asymptomatic, verrucous papules anywhere over the body; when inflamed, they may resemble PG. Molluscum contagiosum are multiple, pearly/skin coloured umbilicated papules; when infected/irritated they may simulate PG. However, all these conditions may be differentiated from PG by histopathology. Complications of PG include ulceration, profuse bleeding, development of satellite lesions or disseminated, eruptive occurrence. Treatment includes curettage with cauterization of the base, surgical excision, sclerotherapy, cryosurgery and pulsed dye laser.

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