Case Report

Atypical Presentation of Sweat Dermatitis with Review of Literature

Abstract

Sweat dermatitis is a peculiar kind of irritant inflammatory dermatoses occurring due to prolonged exposure of retained sweat over the skin. It is commonly seen in hot and dry climates like tropics during summer months due to thermal stress. Typically, parchment paper or cellophane paper like scaling is seen over occluded areas of back, shoulder, and other areas. Here we have reported a varied presentation of sweat dermatitis in the form of its coexistence with miliaria rubra (impending to thermal burn). Further we have also observed co existing pityriasis versicolor and sweat dermatitis where the former has prevented the development of latter. Till date there is very little discussion on this condition, so we have tried to provide a concise review about sweat dermatitis along with its classical to atypical presentation with special emphasis on dermoscopy.

Keywords: *Dermoscopy of sweat dermatitis, heat rash, sweat dermatitis*

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Introduction

India, being a tropical country has a variable temperature ranges at different places. In a metro city of Delhi, average temperature of 44–45°C with low humidity is generally recorded in the months of May and June. Such climatic conditions leads to profuse sweating which is further aggravated by outdoor activities, traveling for longer durations, overcrowding, prolonged working in ill-ventilated places, etc.[1] Sweat has an important role in body temperature regulation but sometimes due to adverse climatic conditions like high air temperature and high humidity, leading to high heat index, the excess sweat produced by the body does not get completely evaporated, and leads to sweat stagnation. Under occlusive conditions this excessive sweat can cause irritant reaction and dermatitis like clinical picture. In the last few months, we have observed many cases of this sweat-induced dermatitis with varied clinical presentations. Out of these, a prototype and 2 atypical cases are described here, and the literature reviewed of this interesting dermatosis.

Case 1

A 6-month-old baby boy was brought to the Dermatology OPD by his mother for brown colored scaly lesions over his back. The rash began as patchy erythema 4 days back which evolved to present state. Mother gave history of wrapping the child completely with multiple layers of clothing to prevent infections. There was no history of atopy.

On cutaneous examination there was brownish "parchment"-like, shiny skin over the entire upper back and shoulder region. Patchy exfoliation was also present [Figure 1].

A diagnosis of resolving sweat dermatitis was made and patient was given emollients. Attendant was advised to keep infant in cool environment and use light and loose cotton clothing. This resulted in rapid improvement.

Case 2

A 40-year-old male laborer presented to dermatology OPD with severe itching and burning sensation along with exfoliation of skin over back for 1 day. He reported history of developing these lesions overnight after sleeping bare bodied over the hot cement flooring of his home. There was no history of any similar rash in the past.

On examination there were well-demarcated maculopapular rashes over entire back, which was beef red in color and studded with multiple pinpoint vesicular eruptions mainly towards the outer/lateral aspect.

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Interspersed between these dew drops like lesions, there were patchy, irregular areas of superficial necrosis. Exfoliation was also present in few areas [Figure 2]. A small elliptical strip of normal skin was preserved in the lower mid back where there was pronounced grooving in lumbar spinal region. Clinical diagnosis of acute irritant reaction to sweat with miliaria rubra was made. Routine hematological and biochemical investigations were normal. On Dermoscopy (Handheld contact dermoscope: DermLite DL3 gen. Polarized view with magnification 20×) there was diffuse background erythema interspersed with yellowish white lacunar areas (dew drop like lesions) probably corresponding to the obstructed sweat gland duct opening. Scattered dark brown colored irregular stellate areas in regions with clinical necrosis were also observed giving a pseudo reticulate appearance along with patchy scaling. This dermocopic finding could be described as "Starry sky" appearance [Figure 3]. Typical white bulls eye sign of miliaria rubra with white globules consisted of a central white area surrounded by a darker halo forming a dot within a dot could also be appreciated [Figure 3a-c].

Biopsy from an area of necrosis showed focal intraepidermal bulla formation, intraepidermal edema and neutrophilic infiltration (epidermal). There was also presence of mononuclear and eosinophilic infiltrate in the dermis. [Figure 4a and b]. Histopathology from miliaria rubra lesion depicted sub-corneal bullae, acantholytic cells along with eosinophilic infiltrate in the dermis [Figure 5].

Final diagnosis of sweat dermatitis was made, and patient was started on predinsolone 30 mg daily which was rapidly tapered and discontinued over a period of 10 days. Topically he was asked to apply cold compresses followed by fusidic acid and betamethasone cream twice daily. Patient responded satisfactorily and skin condition returned to normal in 2 weeks following exfoliation and transient pigmentary alteration.

Case 3

A 23-year-old male working in a corporate office presented with itching and scaling over the back since 6 days. On probing patient gave the history of traveling for 10 hours in a non-air-conditioned bus 7 days back. Cutaneous examination revealed two different types of lesions over the back and shoulders. There were areas of hyper pigmented coarse cigarette paper like scaling present extensively over entire back along with few well demarcated clear areas or apparent islands of normal skin in between [Figure 6 Red arrow]. On dermoscopy (non polarized view) from the pigmented area, there were increased cutaneous markings along with deep brown

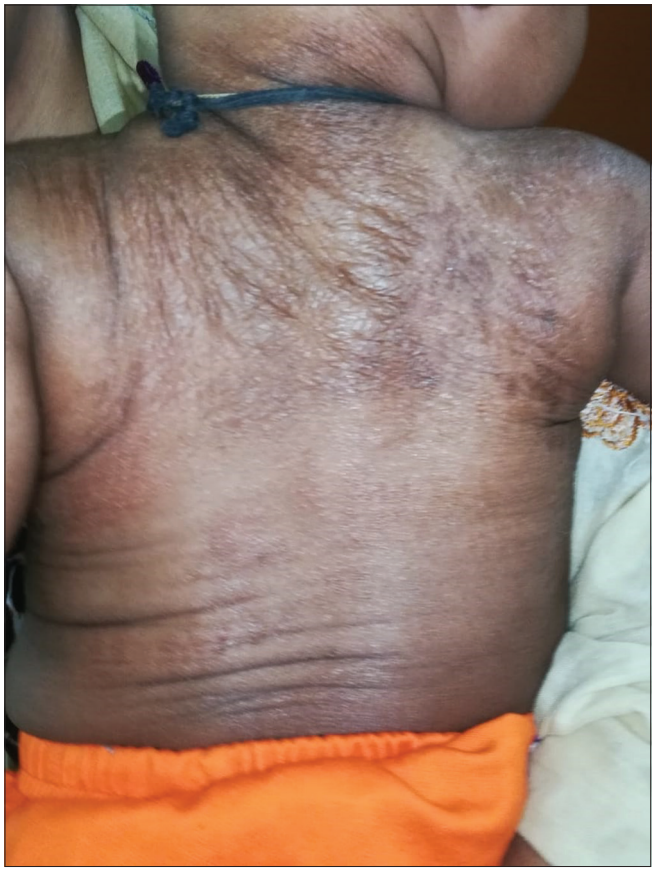

Figure 1: Parchment-like, shiny "crinkled cellophane paper"-like skin over the upper back in a 6 months old child (Case 1)

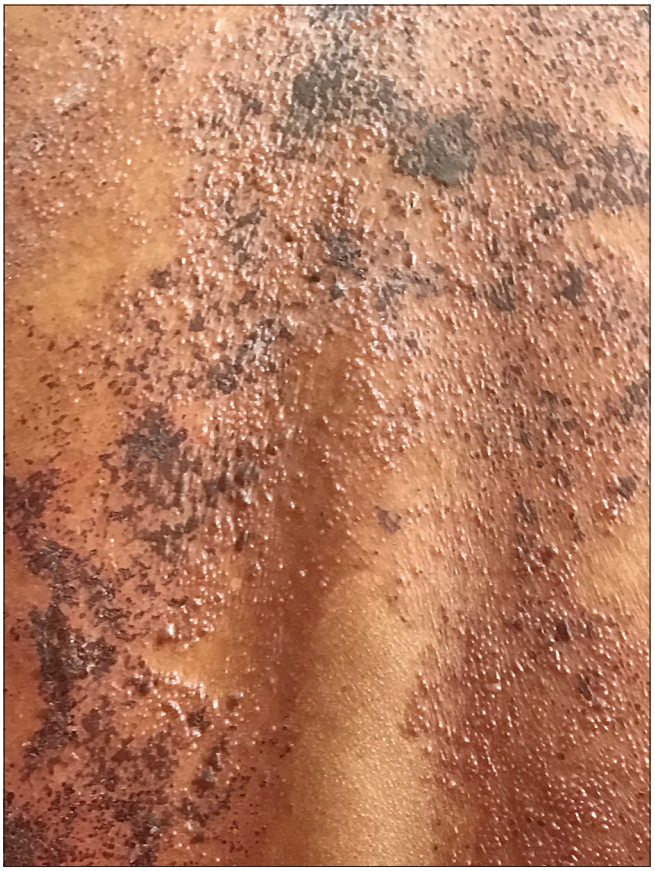

Figure 2: Erythematous maculopapular rash with studded vesicular eruption (miliariarubra) and patchy areas of necrosis. (Case 2)

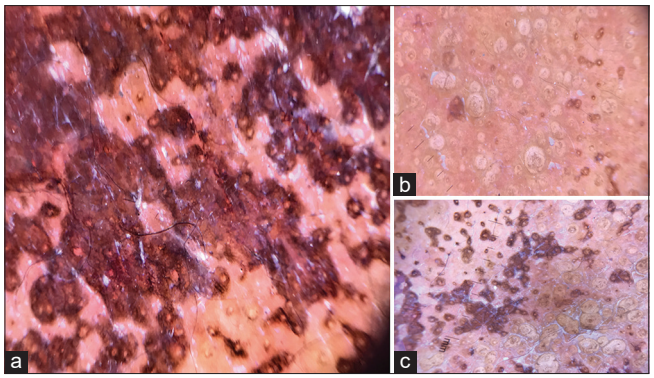

Figure 3: Dermoscopy (Handheld contact dermoscope: DermLiteDL3 gen. (a) Polarized view with magnification 20×) shows yellowish white lacunar areas with scattered specs of necrosis giving a 'starry sky appearance'. (b and c) showing white bull's eye appearance as there is small white dot in bigger dot (lacuna)

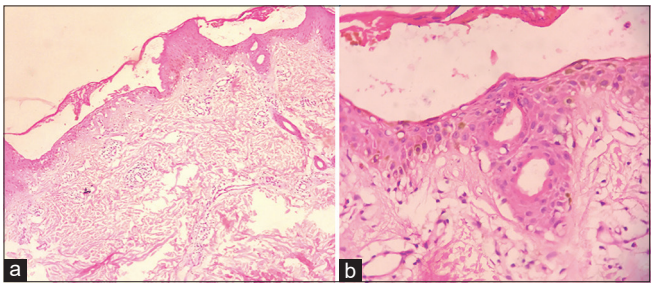

Figure 5: (a) Stain from erythematous papule showing intraepidermal bullae in sub corneal region. (b) (40 × view) H and E Stain from erythematous papule showing intraepidermal bullae in sub corneal region with presence of eosinophilic infiltrate in the dermis. (Case 2) (10 × view) H and E

pigmentary changes with superimposed brownish scales seen at few places [Figure 7].

Secondly, there were well demarcated oval to round hyperpigmented macules on upper back, arms, neck, and extremities having fine powdery perifollicular scales more apparent on tangential viewing. (Figure 6: Blue arrow).

Routine blood investigations were normal. KOH examination was performed on lesions of both morphology, but was positive only from the hyperpigmented macules, showing clusters of yeast cells and long hyphae consistent with pityriasis versicolor (P. versicolor).

A final diagnosis of resolving sweat dermatitis with pityriasis versicolor was made. Patient was started on emollients for sweat dermatitis, tablet fluconazole 400 mg stat and sertaconazole cream once daily application for P. Versicolor lesions. Patient responded well with sweat dermatitis lesions resolving with pigmentary dyschromias.

Discussion

Normal core body temperatures varies between 36°C and 37.5°C (96.8°F and 99.5°F). Sweat glands play an important role in thermoregulation in homeothermic organisms. In hotter months of summer especially in tropics the profuse sweating and subsequent evaporation helps bring down the

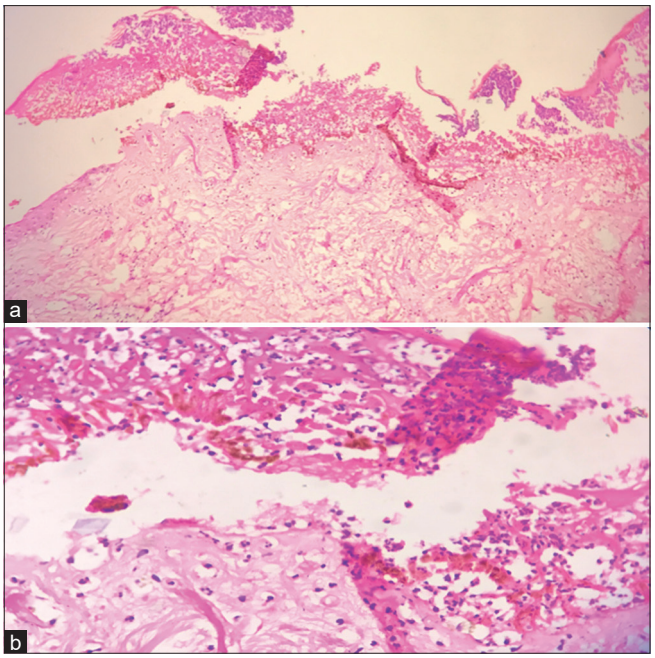

Figure 4: (a) (10 × view) H and E stain from the area of necrosis showing focal bulla formation. (b) (40 × view) H and E stain from the area of necrosis showing intraepidermal edema with neutrophilic infiltration. There is also presence of mononuclear and eosinophilic infiltrate in the dermis. (Case 2)

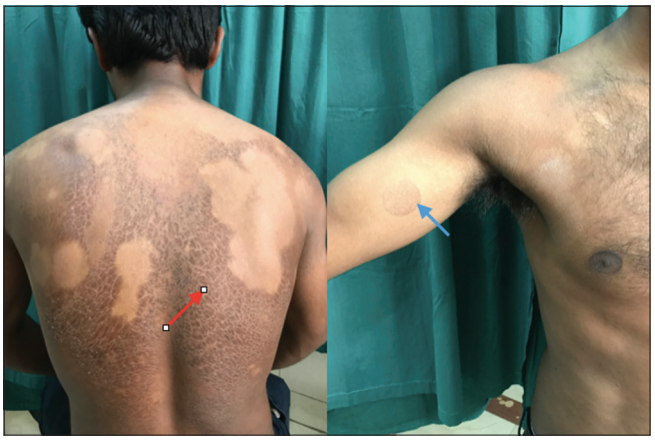

Figure 6: Showing two varieties of lesions. (a) Red arrow-Hyper pigmented coarse cigarette paper like scaling over lower back of sweat dermatitis. (b) Blue arrow-Pityriasis versicolor lesions over shoulder and upper back. (Case 3)

body temperature and keeps the core body temperature in the safe normal range.

Thermal trauma to skin can lead to myriad presentations-burns can occur when skin is exposed to an external heat source with a temperature exceeding 44°C (111°F) for a sufficient period while erythema ab igne ensues when skin is exposed to prolonged and repeated infrared heat insufficient to cause burn.[2] Thermal stress can also at times produce varied pattern of inflammatory dermatosis where sweat has a direct or indirect role in causation. Review of different studies on sweat dermatitis and possible theories causing it has been tabulated below

in Table 1. The term "Frictional sweat dermatitis" was first described by Ramam *et al.* in 1998 where they reported 15 cases with typical friction and sweat induced dermatitis in summer months.[3] Patients presented with burning and stinging sensation together with roughness and scaling where skin was in contact with under surface of undergarments which thus produced a rash with sharp demarcation from normal skin.[3] In 2000 Mehta *et al.*[1] reported similar finding in 380 patients from Thar desert who presented with dark brown charred plaques and roughness of skin over inter scapular, inframammary, axillary folds, waist band, upper chest, and back areas after the episodes of profuse sweating.

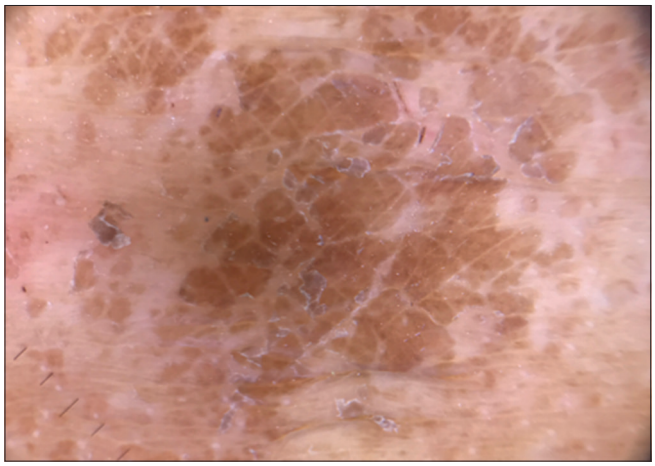

Figure 7: Dermoscopy showing scaling. (Handheld contact dermoscope: DermLite DL3 gen. Polarized view with magnification 20×) (Case 3)

In another study[4] involving 327 patients from same geographical region, the authors reported this scaly dermatosis over areas of skin covered with clothing. Interestingly in all study participants, they found an irritant reaction to patient's own sweat when patch test was performed.

Over the years this type of dermatosis has been reported frequently not only from hot, dry states like New Delhi and Rajasthan but also from hot, humid coastal areas of Puducherry. Gopinath H *et al.*[5] has described morphological spectrum of different sweat-induced dermatosis among which frictional sweat dermatitis constituted 5.3% of patients. Histopathological picture was seen as that of mild spongiform dermatosis. Mehta *et al.* has described dermal mononuclear infiltrates with no sweat gland obstruction at any level in their cases thus differentiating it from miliaria.[1]

Till date various triggering factors have been thought to play a role in causation of sweat dermatitis; most of them emphasize the role of profuse sweating, friction, prolonged exposure of sweat-soaked clothing or undergarment with skin, type of clothing material (tight, porous, synthetic non sweat absorbent), release of unrinsed detergent from clothing,[4] pooling and retention of sweat. Recently Chatterjee and Vasudevan[6] have proposed role of sweat solutes and clothing material in causation of this peculiar dermatitis. They hypothesized; individuals who consumed less amount of fluid and wore traditional porous undergarments were more prone to develop this

Table 1: Review of different studies on sweat dermatitis and possible theories related to it

Study	Clinical setting	Clinical features and investigations	Possible theories of sweat dermatitis
Ramam *et al*.[3] (New Delhi) (1998)	1993-1995. Months of May and June. Temp- 35°C to 45°C. 15 (11 male 4 women)	Mild stinging sensation. Elevated plaque with a glazed, wrinkled surface and sharp margins corresponding to the edges of the undergarment. BX=mild spongiotic dermatitis.	Distribution of lesions below undergarments and other apparel in direct contact with the skin or lower back, against which clothing is pressed when seated, points towards frictional contact dermatitis. Or pressure of tight undergarments also contributes to sweat-soaked undergarment may keep the skin wet for several hours before it drys out or is changed. Sweat acts by leaching out unrinsed detergent from undergarments
Mehta *et al*.[1] (Thar desert) 2000	1993-1995. Months of May and June, Temp-43°C to 47°C. 380 (M: F-3:1)	Presented with dark brown charred plaques. Mild burning or stinging. BX=mild dermal mononuclear infiltrate no sweat gland obstruction	Irritant reaction to stagnant sweat at high temp and low humidity
Chatterjee and Vasudevan.[6] (Thar desert) 2015	2005-2008. Months of May and June. Temp- 43° to 47°C. 327 (M: F-4:1)	Patch test to patients' own sweat showed an irritant reaction in all patients.	Chronic cumulative irritant contact dermatitis to sweat solutes in individuals who consumed less fluid. Irritant reaction to seat solutes occurred in those who wore the traditional porous undergarments which allows the fluid component of the sweat to evaporate easily, leaving the sweat solutes which are in a higher concentration.
Gopinath H *et al*.[5] (Puducherry 2018)	April-June Temp- 23°C to 44°C. 150 pt 8 with sweat dermatitis	Burning, stinging or "tightness" of skin. Dry, hyperpigmented, fissured "parchment" like skin or shiny "crinkled cellophane paper" like skin. BX=mild spongiotic dermatitis	Irritant reaction due to profuse sweating. Role of occlusive clothing and friction negligible.

kind of chronic cumulative irritant contact dermatitis to sweat solutes. Garment made up of permeable material, allowed easy evaporation of water component of sweat, leaving behind higher concentration of sweat solutes, which cause this irritant reaction. In addition, water in the sweat causes water logging which can further leads to disruption of skin barrier function, facilitating delivery of any potential irritant antigens.[7] Sweat also contains other inflammatory mediators including proteolytic enzymes, urea, lactate, histamine, and cytokines such as IL-1 a and IL-8. In addition, the antimicrobial peptides dermicidin and cathelicidin could also act as irritants.[8] All these agents could possibly contribute to development of sweat-induced dermatitis.[8]

In our cases described above, the first case has a presentation similar to previously described cases of sweat dermatitis, including in babies. The excessive covering of infants who have immature sweat ducts commonly cause occlusion of ducts resulting in miliaria. In others, the parchment like skin as in our case, due to irritant dermatitis caused both by water and solute content of sweat may be a manifestation. In the second and third case, however, the manifestations were atypical.

There was a concomitant development of miliaria rubra and sweat dermatitis after an overnight exposure of the bare back to hot cement flooring in Case 2. Gopinath *et al*.[5] described one patient out of their 150 cases who had clinical lesions of sweat dermatitis as linear and parallel ridges and miliaria rubra while other reports on sweat dermatitis do not mention this association. Miliaria is said to develop in un acclimatized or predisposed individuals after several days of exposure to hot and humid environment due to seepage of sweat within layers of epidermis on account of blockage of sweat ducts while sweat dermatitis with superficial cauterization of skin appears within hours of the stagnant sweat irritating the upper layers of the epidermis. It appears that in our case, the initial profuse sweating induced by the hot cement flooring not only directly irritated the skin occluded by the flooring but also could have leached alkaline materials present in the constituents of the cement flooring causing further skin damage bordering on superficial chemical burn. This would have also caused damage to the sweat ducts and other appendages resulting in their occlusion and giving a rough and dry feel to the overlying skin. In addition, the heat retained in the flooring would have continued to induce sweating by the sweat glands which would have leaked intra-epidermally to cause early miliaria formation. The erythema could have been contributed by inflammatory response to both sweat within the skin and sweat mixed with chemicals from without.

Dermoscopy of sweat dermatitis lesions has not been described earlier in literature. In our Case 2, we found miliaria as yellow lacunae "white bulls eye appearance," interspersed in between cauterized skin, so we coined the term "starry sky appearance".

In case 3, the entire back which was in direct contact with clothing and the occlusive seating material which is usually impervious to sweat, developed extensive sweat dermatitis, but surprisingly spared areas involved by P. versicolor. This is to the best of our knowledge not reported before. P. versicolor is a superficial fungal infection which is found as a commensal in several healthy adults but converts into mycelial forms in certain situations including in hot and humid conditions and manifests as scaly perifollicular hyper or hypopigmented lesions mainly over upper chest and back. It can also manifest as pityrosporum folliculitis. Cutaneous lesions morphologically resembling sweat dermatitis caused by Malassezia furfur has not been described in the literature to the best of our knowledge. This excludes the possibility of our case 3 having two different cutaneous manifestations due to a fungal agent. However, it is difficult to offer any convincing explanation as to how this disease offered protection against sweat dermatitis in Case 3.

Park *et al.* have described certain characteristics of skin infected with P. versicolor. The lesional skin was found to have higher hydration content and sebum secretion along with increased Trans-epidermal water loss (TEWL) compared to skin of healthy non-infected adults. The authors believe that the hydrated and sebum rich skin becomes a perfect medium for growth of the organisms which then disrupt the stratum corneum to increase water loss.[9] Whether this increase in TEWL somehow serves as a negative feedback for the regional sweat glands to secrete less sweat thus preventing development of sweat dermatitis in these areas remains to be proven.

On the other hand, Lee *et al.* in their study found the P. versicolor affected skin to be less hydrated than normal surrounding skin along with having increased TEWL.[10] This again indirectly points out to decreased activity of sweat gland function in the regional P. versicolor affected skin which probably explains the atypical presentation in our case.

As elsewhere, the major pattern of sweat dermatitis observed in our outpatient department was like case 1 i.e., parchment paper or cellophane paper like appearance. On dermoscopy (non-polarized view) of these lesions, there were increased cutaneous markings along with patchy, at places linear brownish black pigmentary changes and superimposed scattered whitish scales (case 2).

Conclusions

A wide spectrum of sweat-induced dermatoses is usually seen during the summer months in a tropical country like India, of which the predominant ones are the various types of miliaria. However, a relatively uncommon but probably under reported entity designated "sweat dermatitis" is

being increasingly encountered and added to literature. The unique presentation points to inflammatory response to sweat as a major causal factor in an appropriate background of external occlusion and friction. Fortunately, it responds well and rapidly to measures aimed at reducing sweat secretion besides treating the acute irritant dermatitis. The atypical presentations reflect the complex response of skin to regional variations in sweat gland density, sweat secretion, propensity to sweat duct occlusion, presence of commensal bacteria and yeast and their transformation to pathogenic state. Further observations of similar cases may help in better understanding of this entity and hopefully enable better management.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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