Terra Firma-Forme Dermatosis—More Than Just Dirty

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Introduction

Terra firma-forme dermatosis (TFFD) is a benign disorder that was first described by Duncan, Tschen, and Knox in 1987.[1] The disease is also commonly known as Duncan's dirty dermatosis. The term "terra firma" has Latin roots and is translated to "dry land" in English, owing to the distinct clinical appearance which resembles the Sun-dried clods of land.[2] Often, the condition is misdiagnosed as dermatitis neglecta (DN), contributing to its wide underestimation.

Epidemiology

The estimated prevalence of TFFD according to a retrospective study conducted over a period of 1 year in children under the age of 18 years has been 2.19%.[3] There is no known gender predilection or familial preponderance for TFFD. There is a bimodal peak of incidence with the first peak seen during the first 10 years of life and a second peak during 60–80 years of age.[4-6] TFFD has virtually been reported in all ethnic groups, namely, Asians, Afro-Americans, Hispanics, Middle Eastern, and Caucasians.[5-8]

Pathogenesis

Different authors have laid down various theories in an attempt to explain the complex pathogenesis. One school of thought believes that the nidus of TFFS is retention hyperkeratosis triggered by delayed keratinocyte maturation responsible for impaired desquamation along with an accumulation and dirt and sebum.[9,10] However, Berk *et al*. have proposed that TFFD is largely a papillomatous disorder with acanthosis and orthohyperkeratosis that can trap dirt, sebum, or other environmental debris.[11] According to Erkek *et al*., there is an inhibition of the epidermal keratinoplastic activities which inhibits the detachment of keratinocytes and triggers retention hyperkeratosis and clumping of corneocytes.[12]

The three most commonly cited factors in the etiopathogenesis of DN are the role of microbia, physical factors, and genetic susceptibility with a complex interplay between these agents. Shan *et al*. and Trave *et al*. have postulated the role of *Malassezia* species in the pathogenesis of TFFD. In fact, even saprophytic organisms have been considered as a possible trigger.[13,14]

1. Physical factors

The development of TFFD on sites adjacent to a surgical wound, poor hygiene in physically or mentally disable individuals, over flexures, and underneath a piece of jewelry-clad site, all favor the role of physical factors. Other physical factors incriminated in the pathogenesis of TFFD are prolonged sun exposure, xerosis, sebum, and sweat accumulation.[15,16]

2. *Genetic susceptibility*

A few case reports have suggested the role of genetic predisposition in TFFD; however, the literature on its genetic inheritance is still inconclusive.[4,6] A genetic disorder of delayed keratinization could explain the first peak of TFFD seen in patients between 0 and 10 years of age. Although there is no racial predilection,[17,18] fillagrin gene mutation also plays a minor role.

Clinical features

Classical TFFD is characterized by the development of brownish-gray to black macules and patches having a typical dirt-like appearance [Figure 1]. Other variants include verrucous [Figure 2], papillomatous, and reticular [Figure 3] patterns with islands of spared normal skin between the lesions.[18]

On palpation, often the lesions have a smooth, velvety, or scaly texture with

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fine scales. Often the ailment goes unnoticed owing to its asymptomatic nature. The most commonly involved sites are face, neck, trunk, navel, ankle, and concave contours of body.[19] In pediatric cases, navel is a commonly affected site, where TFFD is often falsely diagnosed as omphalitis or melanocytic nevi.[4] TFFD in postoperative period over surgical sites is also a common presentation.[9] Dermoscopic features include brown scales and large, polygonal brown clods interrupted with furrows and arranged in a linear pattern, creating a mosaic appearance [Figure 4].[2,20]

Histopathology

Histopathology reveals the presence of lamellar hyperkeratosis and retention hyperkeratosis, with focal areas of compact and whorled orthokeratosis, keratin whorls and globules, marked acanthosis, and papillomatosis, along with basal layer hypermelanosis [Figure 5]. The dermis has

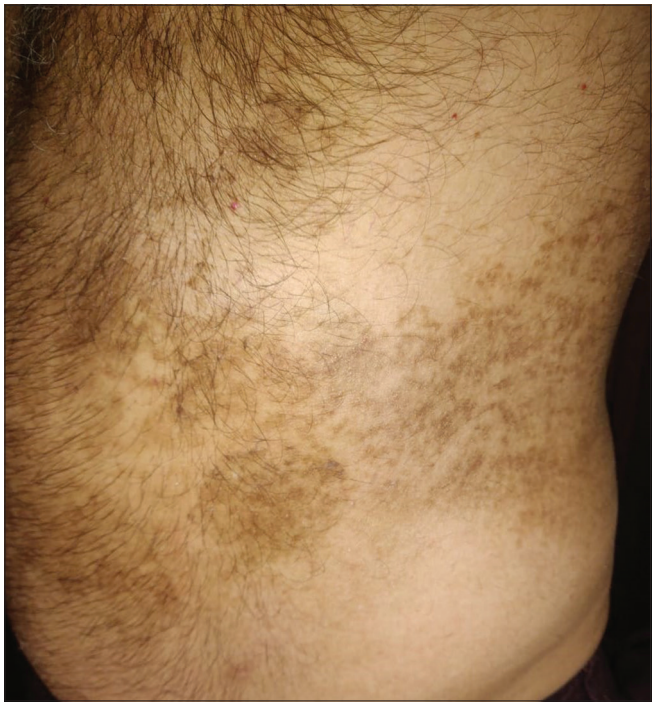

Figure 1: Classical TFFD over flank

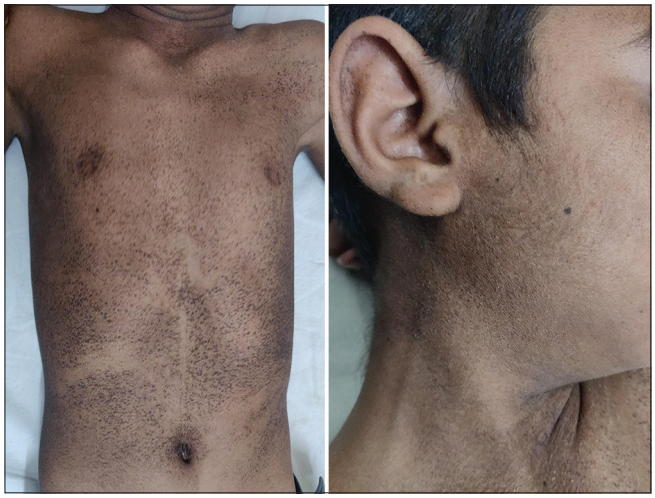

Figure 3: Generalized reticular TFFD in a 10-year-old boy

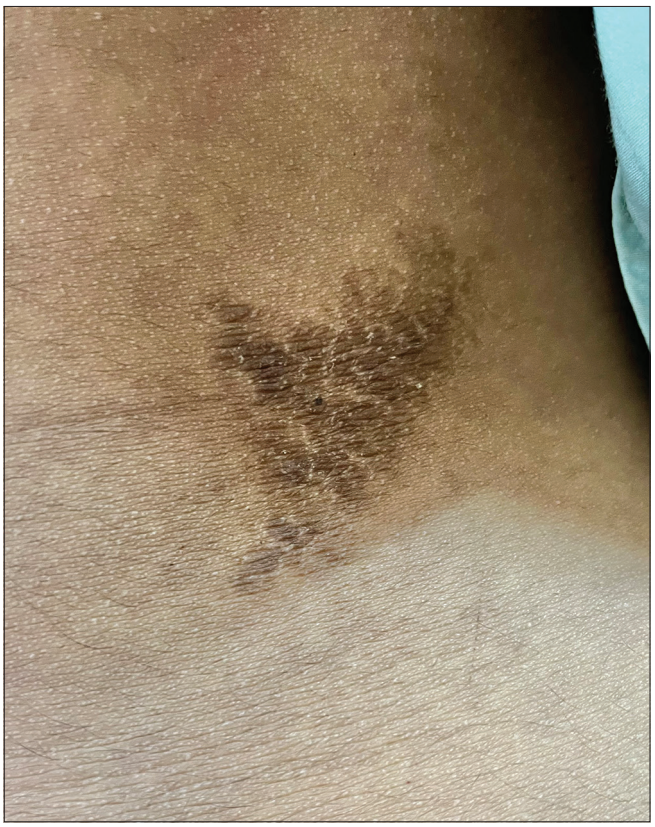

Figure 2: Verrucous TFFD over axillary area

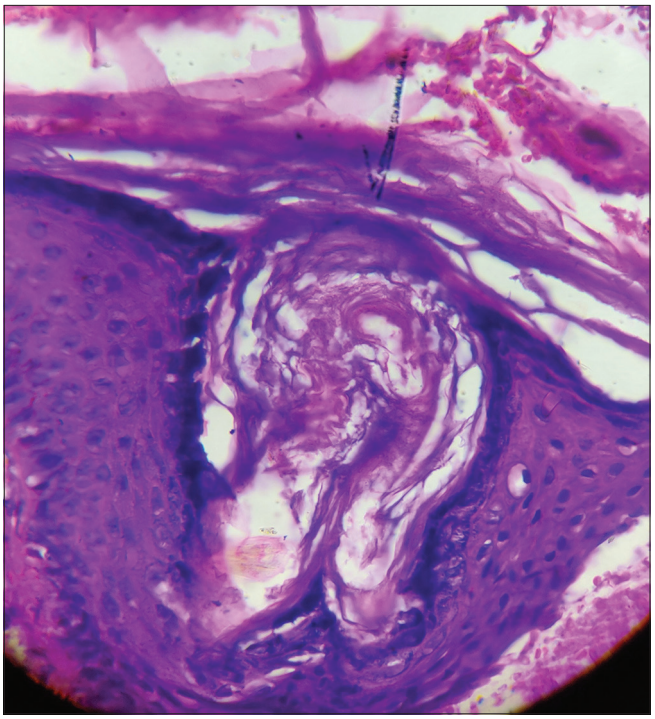

Figure 4: Keratin whorl in the stratum corneum (H and E, 400×)

the presence of mild perivascular lymphocytic infiltration. The periodic acid Schiff stain can reveal yeast aggregates within the whorled orthokeratosis.[15] Unlike DN, there is the absence of anastomosing rete ridges in TFFD.[20]

Differential diagnosis

TFFD is a differential diagnosis for a large number of dermatoses. The most common differential diagnoses in children are DN and the so-called dirty neck sign in atopic dermatitis.[21] Other differential diagnoses include post-inflammatory hyperpigmentation, tinea versicolor, ichthyosis, confluent and reticulated papillomatosis, and acanthosis nigricans.[22-25] Other differential diagnoses have been enlisted in Table 1.

1. Dermatitis neglecta

DN is the most common misdiagnosis of TFFD.[21] Although many authors have highlighted that the differences between these two entities are more semantic than practical, there are still some key differences between these two dermatoses which have been tabulated in Table 2.

The alcohol swab test is one useful test which can differentiate TFFD from DN. While simple soap water can wipe off DN, in order to wipe off the scales of TFFD, only 70% isopropyl alcohol has been found to be effective.

Associated conditions

The most frequently associated disease is atopic dermatitis. Other ailments include xerosis, asthma, allergic rhinitis, and acne. There is also a possible association between TFFD and pregnancy (periareolar lesions), acanthosis nigricans (flexural lesions), insulin resistance, and pituitary adenoma.[2,12]

Figure 5: Dermoscopic images of a case with TFFD showing plate-like scales represented as polygonal clods in mosaic pattern (DermLite DL3, magnification ×10)

Treatment

The usage of isopropyl alcohol in 70% is both diagnostic and therapeutic for TFFD. However, unlike DN, soap-water test is negative [Figure 6]. As TFFD is primarily a disorder of hyperkeratotic retention, keratolytics are used as its first-line treatment. In case of early onset, genital, facial, or generalized TFFD, the patients benefit from a combination of keratolytics and topical calcineurin inhibitors, alpha hydroxy acids, topical retinoids, or salicylic acid. Localized hyperkeratotic TFFD, on the other hand, is better dealt with stronger solvents like acetone or a higher concentration of keratolytics. In fact, recently even fractional CO_2 laser has been utilized as an effective treatment modality by Chun *et al*.[26]

Indian scenario

Despite its wide prevalence, a quick PubMed search reveals that there are only a handful of reports on TFFD from India in the past 8 years [Table 3].[17,27-31] This lack of recognition and research on TFFD in India highlights the need for increased awareness and understanding of this condition within the medical community.

Till date, there has only been one major clinical study on TFFD in India, conducted by Kumar *et al*. The most common clinical pattern in their study was classical dirt-like plaques in 80.43% cases, followed by 6.5% cases of reticular TFFD, and 4.34% cases each of verrucous and linear TFFD.[17]

Conclusion

TFFD is a common but hugely underreported dermatosis with a predilection for the pediatric population. The diagnosis of TFFD in clinically suspected cases can be diagnosed by a positive alcohol swab test but a negative

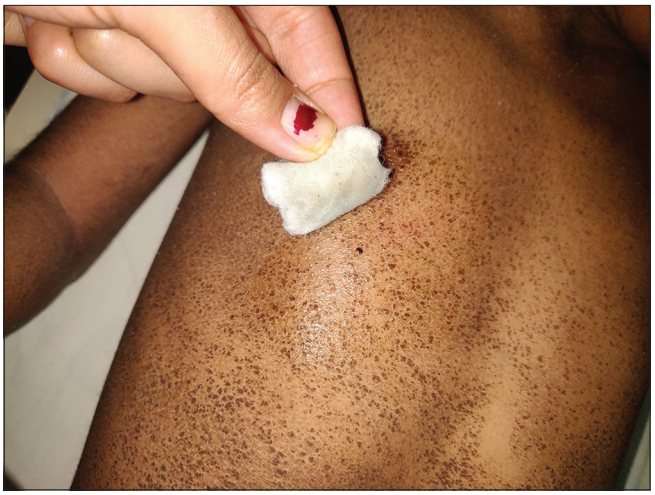

Figure 6: Negative soap-water test

Table 1: Differential diagnosis of TFFD

Diagnosis	Clinical manifestations	Histopathological features	Identifying features
Dermatitis neglecta	Adherent hyperpigmented papules, polygonal plaques, or verrucous lesions with poor hygiene	Hyperkeratosis, acanthosis, papillomatosis, and lamellar orthokeratosis	Hyperpigmented well-define cornflake-like waxy scales on dermoscopy Soap-water test is positive
Acanthosis nigricans	Hyperpigmented velvety plaques and increased skin markings over intertriginous areas	Hyperkeratosis, papillomatosis, minimal to no acanthosis, no hypermelanosis	Dark brownish clods with crista cutis on dermoscopy
Confluent and reticulated papillomatosis	Persistent erythematous to hyperpigmented scaly macules or papillomatous papules and coalescing confluent plaques distributed centrally and exhibiting a reticular pattern over the edges over upper trunk	Hyperkeratosis, papillomatosis with superficial dermal perivascular lymphocytic infiltration	Cobblestone pattern formed by polygonal flat-topped globules surrounded by whitish striae on dermoscopy
Dirty neck syndrome	Reticulate or rippled hyperpigmentation in patients with atopic dermatitis, predominantly involving the anterolateral aspects of the neck	Acanthosis, basement membrane thickening, epidermal hypermelanosis, dermal melanophages	Presence of other clinical features of atopic dermatitis
Postinflammatory hyperpigmentation	Hyperpigmented macules or patches over sites of healed inflammation	Epidermal or dermal melanosis	
Icthyosis	Genodermatoses with impairment in keratinization	Icthyosis vulgaris: moderate hyperkeratosis hypogranulosis X-linked icthyosis: hyperkeratosis with hypergranulosis	Positive family history
Intertriginous granular parakeratosis	Hyperpigmented hyperkeratotic papules and coalescing plaques over intertriginous areas	Acanthosis, hyperparakeratosis and papillomatosis, with basophilic keratohyalin granules	
Verrucous epidermal nevus	Congenital to early-acquired fleshy-to-brown, papillomatous papules and coalescing plaques, along Blaschko's lines	Acanthosis, hyperkeratosis, papillomatosis, and epidermolytic hyperkeratosis	Thick brown branched lines and circles, terminal hairs with dotted vessels on dermoscopy
Frictional melanosis	Hyperpigmented patches over bony sites and friction-prone regions	Epidermal hypermelanosis, dermal melanophages	Brownish structureless zones in reticular pattern on dermoscopy
Macular amyloidosis	Pruritic hyperpigmented patches in ripple-like patterns over extremities and upper back	Basal layer hyperpigmentation. Globular deposits of amyloid material in papillary dermis and melanophages	Central brown to white hub at the center with brown fine radiating streaks, leafy projections, and dots on dermoscopy
Tinea versicolor (pigmented pityriasis versicolor)	Discreet to coalescent brown, hyperkeratotic scaly patches over head and neck	Orthohyperkeratosis and budding spores in stratum corneum with silent dermis	Yellow-green fluorescence on wood lamp
Ashy dermatosis	Round to oval ashy-colored hyperpigmented macules symmetrically involving the trunk	Lichenoid infiltration with macrophages	Irregular brown-grey linear dots and globules over pinkish-brown background. Irregular linear vascular pattern on dermoscopy
Dowling–Degos syndrome	Genodermatosis with macular or papular folliculocentric lesions. Arrangement of lesion in a lacy or reticular pattern predominantly over folds	Elongated or branched rete ridges having increased melanin deposition over the tips. Suprapapillary epithelium sparing	Irregular brownish projections around central hypopigmented, indicative of follicular epithelium on dermoscopy
Acroangiodermatitis of Mali	Purplish-blue to brown papules and plaques located on the medial and lateral malleoli	Hyperparakeratosis, epidermal hyperplasia, pseudo-sarcomatous capillary proliferation, and perivascular inflammation in the dermis, with extravasated red blood cells and hemosiderin deposits	Red or blue lacunae, white rail lines, with whitish veil along with scattered hemorrhagic crusts over a pinkish background on dermoscopy

Contd...

Table 1: Contd...

Diagnosis	Clinical manifestations	Histopathological features	Identifying features
Epidermolytic hyperkeratosis of the nipple and areola	Warty, hyperpigmented papules or plaques on the nipple and/or areola	Orthohyperkeratosis, filiform acanthosis, papillomatosis, and keratin plugging, hyperpigmented rete ridges, and a dermal lymphocytic infiltrate	Whitish papillomatous surface with white scales and brown hyperkeratotic deposits distributed on the cristae on dermoscopy
Scalp discoloration from selenium sulfide shampoo	Red-brown discoloration of scalp skin and appendages with/without desquamation	Pigment deposit within keratotic debris and parahyperkeratosis	Suggestive clinical history

Table 2: Differentiating features between terra firma-forme dermatosis and dermatitis neglecta

	Terra firma-forme dermatosis	Dermatitis neglecta
Synonym	Duncan's dirty dermatosis	Dermatitis passivata
Etiology	Unknown Genetic predisposition	An acquired disorder in patients with unclean habits
Pathogenesis	Delayed maturation and retention of corneocytes and melanocytes	Accumulation of sebum, sweat, and dirt within the keratin layers
Hygiene status	Good Adequate cleansing	Poor Inadequate skin cleansing
Clinical features	Brown grey velvety patches and plaques. Verrucous, papillomatous, or reticular patterns with islands of sparing	Hyperpigmented macules and plaques with corn-flake-like brown scales
Histopathological feature	Lamellar hyperkeratosis, whorled orthokeratosis, keratin whorls, marked acanthosis, basal layer hypomelanosis, papillomatosis, and perivascular lymphohistiocytic infiltration	Orthokeratotic hyperkeratosis and mild acanthosis with anastomosing rete ridges
Dermatoscopic features	Hexagonal brown plate-like scales, which are organized in cobblestone or mosaic patterns	Waxy cornflake-like brownish scales
Soap-water test	Wiping with soap-water does not clear the lesions	Wiping with soap-water clears the lesions
The alcohol swab test	70% isopropyl alcohol swab clears lesions	70% isopropyl alcohol swab clears lesions
Management	70% isopropyl alcohol wipe, emollients, keratolytics, alpha-hydroxy acids, topical retinoids, and CO_2 laser (for resistant cases)	Regular bathing and good hygiene practice Keratolytics for recurrent cases
Prognosis	Poor response to treatment and frequent recurrences	Good response to treatment

Table 3: Reports on TFFD from the Indian subcontinent

Authors	Type of report	Clinical features
Kumar *et al*.[17]	Observational study	46 cases; Classical dirt-like plaques (80.43%), reticulate (6.5%), verrucous (4.34%), stuck-on appearance (4.34%), and linear lesions (4.34%)
Panchal *et al*.[28]	Case report	19-year-old female; extensive TFFD with symmetrical brownish-black verrucous papules and plaques over the chest and medial aspect of thighs
Naveen *et al*.[29]	Case report	20-year-old male; multiple hyperpigmented plaques of TFFD over healing lesions of chicken pox on the trunk and back.
Verma *et al*.[30]	Case report	40-year-old male; on treatment for psoriasis. TFFD following erratic application of heavy emollient and betamethasone valerate-salicylic acid ointment over bilateral legs. Dermatoscopic features: cobblestone appearance.
Saha *et al*.[31]	Case report	8-year-old boy; TFFD lesions with hyperkeratotic papules over neck and upper chest.

soap-water test. A consistent finding in histopathology is whorled hyperkeratosis. Noninvasive modalities like dermoscopy could aid in the rapid diagnosis, averting the need for histopathological analysis. A dermatologist must always be on the lookout to proficiently differentiate TFFD from other similar-looking conditions.

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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