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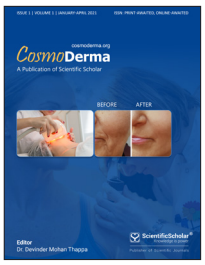

Focus

Trichostasis spinulosa: An entity with cosmetic concern

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INTRODUCTION

The high prevalence of trichostasis spinulosa (TS) contrasts with the relative dearth of recognition paid to it; TS is often dismissed as a cosmetic entity. Moreover, the paucity of reports indicates that TS is indeed underrecognized.[1] Treatment of TS continues to be a challenge to this day, due to the scarcity of high-quality evidence. Suboptimal therapeutic regimens have been guided largely by case reports, with the mainstay of treatment being keratolytics and topical retinoids.[2] Recently, lasers have emerged as a promising treatment modality.[2,3] We present a review of the pathogenesis, clinicopathologic features, associated conditions, dermoscopic features, and treatment of TS.

HISTORY

TS is a distinct, benign, pilosebaceous unit disorder, stemming from the entrapment of bundle of tiny vellus hairs encircled by a keratinous sheath in a dilated follicular infundibulum.[4] This unique disorder was first described in 1901 by the German dermatologist, Felix Franke under the title of "das pincelhaar ("pincelhaar meaning paintbrush hair) and "thysanothrix" (thysanos – meaning fringe). Thereafter, different terminologies have been used such as "Lanugo Komedonen", "Keratosis spinulosa cum trichostasi", "Dysplasia pilorum thysanoformis" till the time Noble coined the term "TS" in 1913.[5]

PATHOGENESIS

The pathogenesis of TS is not completely elucidated. Chief pathology lies in successive production and retention of vellus telogen club hairs from a single hair matrix in a pilosebaceous follicle.

One hypothesis proposes that disturbance in the dynamics of hair papilla and subsequent exaggerated papillary cyclical activity is the cause for the growth of vellus hairs within the follicle.[6] Another hypothesis proposes that abnormal keratinization of follicular epithelium and the resulting keratotic plugging caused by internal (congenital dysplasia of hair follicles) or external factors (occlusive preparations such as oils, dust, heat, ultraviolet light, and industrial irritants) might damage the follicles and prevent the extrusion of normal hair, leading to their retention.[6,7]

Long-term topical corticosteroids are thought to instigate changes of TS by cornification of the pilosebaceous duct and induction of hypertrichosis through the stimulation of vellus hair

growth.[8] Terminal hair TS following local application of minoxidil is reported, minoxidil-induced transformation of retained vellus to terminal hair is believed to be the reason.[9]

In addition to the above theories, actinic skin damage, aging and abnormal angulation of isthmus at the level of infundibulum have all been postulated to a play a role. Increased prevalence of *Cutibacterium acnes* (formerly known as *Propionibacterium acnes*) and *Pityrosporum* species is reported by some authors.[10] They have suggested that there is a possible provocating and aggravating role of these microbes in induction of follicular keratosis and favoured microbial eradication for better results. However, studies in this respect failed to deliver any conclusive evidence.

TS is described in association with space-occupying lesions that constrict follicle infundibulae by tumor cells or tumor-related fibrosis,[11] such as melanocytic nevi,[12] collagenoma,[13] seborrheic keratosis,[14] syringomas,[14] follicular sebaceomas (trichofolliculomas), or nodular basal cell carcinomas.[15,16]

CLINICAL FEATURES

TS is a fairly common disorder than is presumed. However, most often it is underdiagnosed and under-reported owing to the usual lack of any subjective symptoms, rather than its rarity.

Primary TS presents as isolated finding, spotted only as an incidental discovery in cases attending for unrelated skin problems.[15] Less commonly, it is observed in association or found to develop within other cutaneous conditions as mentioned above (secondary TS).[7,17] The onset is usually after adolescence, elderly being most affected perhaps due to prolonged actinic damage. Childhood cases are rare.

Sexual predilection is absent though females are more concerned and seek treatment. TS preferential involves body areas with abundant sun exposure and pilosebaceous units, commonly the face [Figure 1], neck, chest, upper arms, and interscapular regions [Figure 2].[4,18] Truncal distribution of asymptomatic lesions is thought to be caused by continuous pressure of garments.[8]

In the areas of affection, TS manifests as minimally keratotic plugs that either project over the follicular orifices containing central, 5–80, dark short hairs (resembling the bristles of paintbrush) or seen to be embedded within the orifices as small comedo-like dots. Hence, they are mistaken for blackheads by the patients.[19,20] Normal appearing follicles are found interspersed with those containing the hyperkeratotic spinules in some cases while in others all the follicles of the involved area are plugged.

Patients rarely complain of the roughness of the face, scaling, or pruritus. Erythema, edema, and secondary pustulation are noted around lesions that have been manipulated to express the keratotic material. Psychological distress from the appearance of the lesions usually prompts patients to seek care.[18]

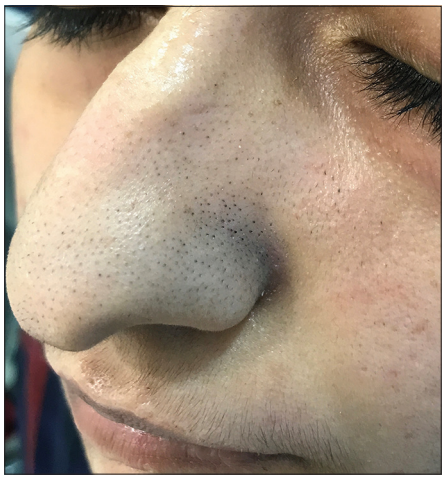

Figure 1: Clinical presentation of trichostasis spinulosa on the nasal dorsum and ala nasii as dark dots.

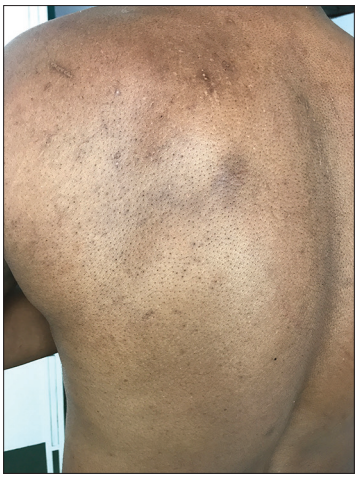

Figure 2: Trichostasis spinulosa affecting back and presenting as minimally keratotic follicular paupules with multiple hair bristles.

Two variants are defined that are differentiated based on the age of onset, anatomic distribution, and the presence or absence of pruritus.[21] The classical variant is the non-pruritic type which is frequently seen in the elderly in a localized distribution, commonly affecting the tip of the nose, ala nasi, chin, cheeks, face, or the interscapular region.[21] The pruritic type seen in young adults is the second variant and is characterized by widespread distribution, extra-facial involvement, and in association with other follicular hyperkeratotic disorders, chronic renal failure, and benign cutaneous tumors.[15,21,22]

Uncommon presentations include involvement of scalp mimicking black dots of alopecia areata,[11,23] involvement of

axilla,[24] lower eyelid,[7] nevoid pattern[25], and generalized distribution in a case with chronic renal failure.[22]

DIFFERENTIAL DIAGNOSIS

Dermatoses presenting as keratotic follicular papules may be considered in the differential diagnosis of TS. These include keratosis pilaris, lichen spinulosus, open comedones of acne, eruptive vellus hair cysts, ichthyosis follicularis, hypovitaminosis A, hypovitaminosis C, dyskeratosis follicularis, Kyrle's disease, keratosis pilaris atrophicans faciei, atrichia with papular lesions, keratosis pilaris decalvans, pityriasis rubra pilaris.[9,21] Pili multigemini, popularly known as "compound hairs" is another close differential and is characterized by the presence of bifurcated or multiple divided hair matrices and papillae in contrast to single papillae of TS, giving rise to the formation of multiple hair shafts within the individual follicles.[9,26] Of the above list, acne comedones, lichen spinulosus, keratosis pilaris, and eruptive vellus hair cysts very closely simulate TS. Distinguishing clinical, histological, and dermoscopic features of these entities are summarized in [Table 1].[18,19,27-32]

DIAGNOSIS

TS is often missed upon gross naked eye examination. Close observation with a handheld magnifying lens shows dark grouped hair bristles projecting from the horny follicular plugs.

DERMOSCOPIC EXAMINATION

Dermoscopy enables accurate rapid diagnosis of TS and also aids in monitoring response to treatment.[17] The dermoscopic findings are classified as signs of hair retention seen as vellus hairs and tufts of hairs [Figure 3] and signs of follicular keratoses described as dermoscopic black dots [Figure 4] and orangish-yellow plugs occupying different sizes of dilated pores. Slight compression of the lesions as in immersion contact dermoscopy helps separate and spread the hairs, enabling visualization of each hair shaft tip.[33,34] Dermoscopic differential diagnosis is elaborated in Table 1.

HAIR MOUNT AND TRICHOGRAM

Microscopic analysis makes the diagnosis easy. Mounting the follicular contents expressed by gentle manual pressure or hair pull or extraction by a pair of forceps, tweezers or comedone extractor typically reveals a bunch of parallelly arranged more than 6 vellus hairs glued together by a gelatinous material in the midportion, the clear greasy substance does not extend to the distal or proximal ends of the hairs and leaves a portion of them uncovered at both ends [Figure 5]. These retained hairs are usually more pigmented and thinner at the distal end, the proximal intrafollicular portion is lighter in color and bulbous indicating that they are in the telogen phase of the hair cycle.[6,35]

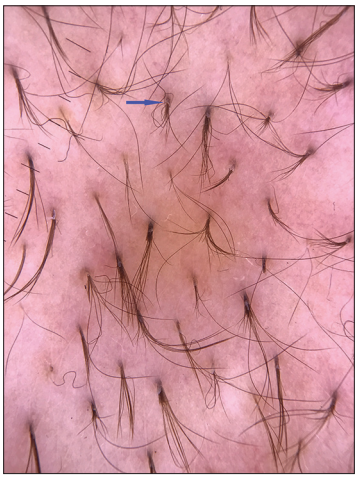

Figure 3: Dermoscopic examination (Dermlite DL4N, polarised mode, x10). Hair retention seen as tufts of tiny dark hair emerging from follicular ostia.

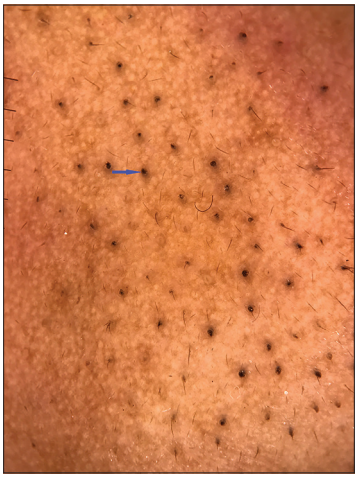

Figure 4: Dermoscopic examination (Dermlite DL4N, polarised mode, x10). Follicular keratosis seen as dermoscopic blackheads.

HISTOLOPATHOLOGICAL EXAMINATION

A punch biopsy is usually not indicated or necessary to establish a diagnosis of TS. Histologic features of TS include acanthosis and hyperkeratosis of perifollicular epithelium, mild inflammatory infiltrates, multiple thin vellus hair shafts, and increased amount of lamellar keratin inside the infundibular region of follicles, corresponding to the retained hairs and keratin plug, respectively.[6,10,26]

TREATMENT

TS, while otherwise being medically harmless, runs a chronic refractory course with lesions usually reappearing after treatment discontinuation.[1]

Table 1: Differential Diagnosis of Trichostasis Spinulosa.

Characteristic	Trichostasis spinulosa	Acne open comedones	Keratosis pilaris	Eruptive vellus hair cysts	Lichen spinulosus
Anatomical distribution Common Others	Face, especially the nose, neck, chin, interscapular region[18] Chest, back, especially abdomen and extremities, scalp	Face, back Neck, upper chest, shoulders	Extensor and lateral aspects of the proximal extremities, cheeks. Neck, torso, and buttocks[28,32]	Chest, Sternal area, abdomen Anterior flexures of limbs, trunk, buttocks forehead and face	Trunk, extensor surfaces of the upper extremities Neck, buttocks, abdomen, knees cheeks[32]
Age	All age, mostly elderly[29]	All ages, mostly adolescents	All ages, most often early childhood[29]	Children and young adults[29]	Children and adolescents[18]
Sex	No predilection	Males slightly more affected	No predilection	No predilection	Male preponderance
Clinical features	Horny plugs within dilated follicles with protruding hairs Comedo like impacted plugs[28,29]	Solid, horny impactions that distend hair follicle[18]	Spiny grouped/ scattered keratotic papules with subtle perifollicular erythema[28,32]	1 to 5 mm soft to firm skin coloured or hyperpigmented, dome-shaped papules topped with central puncta, umbilicated or a hyperkeratotic crust[28,30]	Skin coloured asymptomatic or mildly pruritic follicular spiny papules in patches[18]
Dislodgement of keratotic plug	Effortlessly dislodged[29]	Difficult to dislodge	Effortlessly dislodged[29]	Removal not possible due to dermal location of pathology[29]	Removed with difficulty
Microscopic analysis/ Hair mount of removed follicular plug	Bunch of vellus hairs enclosed by a keratinous sheath[28,29]	Very few three to six vellus hairs per comedone are sometimes present and are usually coiled within the horny plug[17]	Single coiled vellus hair surrounded by keratin material[15,28,29]	Not possible as plug cannot be removed[28,29]	Keratinous material with no hairs
Histology	Acanthotic epidermis enclosing a dilated follicle containing several vellus hairs glued together by a keratin plug. Perifollicular infiltrate and infiltrate around arrector pili muscle may be seen[28,29]	Plugging of the pilosebaceous orifice by sebum on the skin surface[34]	Dilated follicle with single coiled hair, infundibular plugging mild perifollicular inflammation and fibrosis[28,29]	Cystic spaces lined by stratified squamous epithelium containing variable quantity of laminated keratin and several vellus hair located in mid or upper dermis[28,30]	Dilated follicular ostia with compact hyperkeratosis, focally associated with a perifollicular and perivascular lichenoid infiltrate[33]
Dermoscopy	Vellus hairs, tufts of hairs Dermoscopic black dots Yellowish orange horny plugs – follicular keratosis[28]	Brown- Yellow hard central plug[30]	Mostly single, sometimes 2 or 3 vellus hairs that are frequently coiled, semi-circular or looped, peri-follicular erythema, peri-pilar casts, and vascular ectasias. Pigmented globules suggest postinflammatory hyperpigmentation[28,32]	1. Brownish, round-to-oval structures - cysts with laminated keratin 2. Brownish halo-inflammation 3. Central/eccentric pores – opening of cysts into the epidermis 4. Blue homogenous areas- Tyndall effect of keratin 5. Peripherally arranged radial capillaries (may or may not be present)[28,30]	Round-to-oval yellowish areas with keratotic follicular plugs surrounded by mild erythema

(*Contd...*)

Table 1: (*Continued*)

Characteristic	Trichostasis spinulosa	Acne open comedones	Keratosis pilaris	Eruptive vellus hair cysts	Lichen spinulosus
Treatment	Keratolytics, tretinoin not effective Treatment-resistant Long remission possible with lasers[28][29]	Topical retinoids, Azelaic acid Benzoyl peroxide Salicylic acid and glycolic acid peels[17]	Keratolytics, topical retinoids effective Oral retinoids and lasers like PDL, KTPL, alexandrite laser, long-pulsed diode laser, Q-switched Nd:YAG laser, and fractional carbon dioxide laser are effective[28,29]	Spontaneous resolution via transepidermal elimination may occur Needle evacuation, topical and oral retinoids, dermabrasion and lasers[28,29]	Spontaneous resolution may be seen Keratolytics Topical and oral retinoids Vitamin D analogous Sometimes persistent with relapsing course[32]

Figure 5: Hair mount of the plucked hairs showing multiple vellus hairs bundled together by a keratinous sheath with characteristic paintbrush appearance.

The existing treatment modalities that have been explored with varying success range from simple minimally invasive office-based procedures such as needle evacuation, hydroactive adhesive tape stripping, keratolytics to laser and energy based interventions.[1,18]

Needle evacuation and extraction with the help of forceps, tweezers, or comedone extractor is painful and can be complicated by scarring and recurrences.[6,36,37] A modification of cyanoacrylate skin surface stripping (used to harvest a thin layer of stratum corneum) was also described to mechanically debride the spiny projections. A drop of cyanoacrylate liquid adhesive is placed on a transparent glass slide, which is then pressed firmly onto the target site of the skin for 15–30 s. The horny plugs along with the hairs of TS get attached and subsequently removed when the slide is rocked from side to side with little force. Erosions and oozing can occur with this method.[37]

Depilatory wax and removal of hairs by depilatory cellulose lacquer provide temporary relief.[6] Hydroactive adhesive tape (Biore), a deep cleaning pad can be applied on wet skin for 10 min and then carefully peeled off. It contains a cationic (positively charged) hydrocolloid with active agent polyquaternium 37 that binds to comedonal plugs which are rich in acidic amino acids (negatively charged). Instant removal of few plugs with hairs can be noticed.[1,38]

KERATOLYTICS

Keratolytics like lactic acid, urea, ammonium lactate, salicylic acid, Whitfield's ointment have all been tried and found to be only temporarily and minimally effective.[6] Favorable results were seen when used after depilatory agents.[19] Salicylism, contact dermatitis, postinflammatory pigmentation are the rare probable side effects.

TOPICAL RETINOIDS

Tretinoin 0.025%, 0.05%, and adapalene 0.1% by virtue of their comedolytic, antiproliferative, anti-inflammatory properties normalize the defective epidermal turnover, correct the hyperkeratosis and relieve the obstruction in TS.[6] Adapalene, when compared to tretinoin causes less irritation and is better tolerated. Higher concentrations of tretinoin and tazarotene may be used for extra facial lesions.

CHEMICAL PEELS

Repeated capryloyl salicylic acid peelings at concentrations between 20% and 30% decrease sebum secretion, disrupts intercorneocyte cohesion, leading to desquamation and good improvement of TS. Its lipophilic nature permits better penetration up to the mid-portion of the follicular canal where excessive keratinization, the initial event in keratotic plug formation, takes place.[39] Glycolic acid and Jessner's peels known to be effective in treating other follicular keratosis,[40] can be given a try.

LASER HAIR REMOVAL

Selective permanent destruction of pigmented hair using lasers (694-nm ruby laser, 755-nm alexandrite, 810-nm

diode lasers, long pulse1064-nm ND: YAG laser) is found to be safe and successful in slowing hair regrowth in TS, offering a definitive curative option.[3,2,40-43] Improvement in the skin texture, appreciated as reduction in roughness is an added cosmetic advantage besides complete clearance of the comedo-like plugs. Areas with thinner skin such as the face and axilla allow better penetration and delivery of laser energy and respond better than back and extremities. The best long-term results are achieved with the alexandrite and diode hair removal lasers with no statistically significant difference in efficacy and tolerability between the two.[3,42]

Anagen hairs are chiefly amenable to laser ablation. TS, as is a disorder resulting from retention of telogen hairs may not respond comparable with terminal hairs and there is a possibility of recurrence of lesions requiring longer periods of follow-up.[2,3,43] Nevertheless, data on laser type, device parameters, number and interval between sessions and follow-up period for any recurrence is lacking.

CONCLUSION

TS is a common though underrecognised entity which can be easily mistaken when the distribution is atypical. Careful examination with dermoscopic evaluation is necessary for the correct diagnosis. Future large randomized control trials are required to compare laser modalities with each other and combination therapies with non-laser options, and to establish optimal treatment protocols.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflict of interest

There are no conflict of interest.

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