Original Article

Cutaneous Manifestations of Obesity and Their Correlation with BMI and Metabolic Comorbidities: A Cross-Sectional Study from a Tertiary Care Hospital in South India

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Abstract

Background: Obesity produces distinctive skin changes driven by insulin resistance, inflammation, friction, and venous stasis. Regional data from South Indian tertiary care are limited. The objective is to map cutaneous disorders in obese adults, test gradients across obesity classes, and examine co-occurring metabolic comorbidities. **Material and Methods:** Standardized head-to-toe examination with adjunct tests (KOH, Wood's lamp, dermoscopy) and condition-specific grading (e.g., Burke for acanthosis; CEAP for venous disease). Anthropometry and blood pressure were recorded; fasting glucose, HbA1c, and lipid profile obtained where feasible. Metabolic syndrome followed IDF-2005 (South Asian cut-offs). Group comparisons used χ^2 (including trend) and ANOVA/Kruskal-Wallis; Poisson regression with robust variance adjusted for age and sex. **Results:** Acanthosis nigricans led the spectrum, followed by acrochordons and striae. Infective dermatoses were frequent, predominantly dermatophytosis. Psoriasis, frictional hyperpigmentation, plantar hyperkeratosis, chronic venous insufficiency, and a small pigmentary cluster were observed. Frictional hyperpigmentation and plantar hyperkeratosis showed dose-response increases across obesity classes; psoriasis trended upward but was imprecise due to a very small severe-obesity cell. Recorded comorbidities included hypertension, diabetes, dyslipidaemia, polycystic ovarian disease, and hypothyroidism. **Conclusion:** Obese adults exhibited a broad, BMI-linked cutaneous profile with infectious, frictional, and vascular signals. Readily recognised lesions acanthosis, multiple tags, plantar hyperkeratosis, patterned hyperpigmentation, intertrigo should prompt streamlined screening for blood pressure, glycaemia, lipids, and central adiposity. Larger prospective, multi-centre studies with protocolized metabolic phenotyping are warranted.

Keywords: Obesity; cutaneous manifestations; acanthosis nigricans; acrochordons; dermatophytosis; metabolic comorbidities; chronic venous insufficiency; South India; cross-sectional study.

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Introduction

Obesity reshapes the skin as surely as it burdens the heart and liver, and dermatology offers a quick, visible readout of that internal strain. Across narrative syntheses, excess adiposity, insulin resistance, and low-grade inflammation converge on cutaneous endpoints acanthosis nigricans, acrochordons, intertrigo, infections, keratinization disorders, impaired healing, and venous changes linking bedside signs to cardiometabolic risk.[1–5] Indian clinic-based data echo this pattern. In a 300-patient cross-section, obesity grades tracked stepwise rises in acanthosis nigricans, skin tags, plantar hyperkeratosis, and superficial fungal disease, positioning these findings as practical "flags" for metabolic stress.[6] A Central-West Indian series added metabolic texture high rates of insulin resistance, diabetes, and atherogenic lipids while again identifying acanthosis nigricans, tinea, and acrochordons as the leading dermatologic companions of elevated BMI and waist circumference.[7] Case-control work from Nepal further showed that overweight/obesity independently associates with dermatophyte infections, acanthosis, and skin tags, with frequency rising by obesity class and persisting after adjustment.[8]

Beyond these staple lesions, several conditions broaden the cardio-cutaneous bridge. Periorbital melanosis carried higher fasting insulin, HOMA-IR, and an adverse leptin-adiponectin profile independent of BMI, suggesting an accessible cue to interrogate insulin resistance.[9] Hidradenitis suppurativa cohorts demonstrated heavier metabolic loads greater BMI, blood pressure, fasting glucose/insulin, and markedly higher metabolic-syndrome prevalence, supporting routine screening even when HS severity is modest.[10] In paediatrics, atopic

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dermatitis clustered with central adiposity, elevated triglycerides and blood pressure, and a sharply higher odds of metabolic syndrome, hinting that metabolic risk can accumulate early when inflammatory skin disease coexists.[11] Venous pathology also intersects with weight: among adults with chronic venous insufficiency, higher BMI correlated with advanced CEAP grades and duplex-confirmed reflux, aligning obesity with stasis-related skin change and ulceration risk.[12] In the diabetes clinic, infectious dermatoses predominantly candidal and dermatophyte were common and tracked with poorer glycemic control, underscoring how hyperglycemia amplifies cutaneous vulnerability.[13]

Psoriasis exemplifies the bidirectional ties between skin and metabolism: populations with psoriasis show excess obesity, insulin resistance, dyslipidemia, NAFLD, and cardiovascular events, with severity often scaling risk; weight loss improves cutaneous control and therapeutic response, while biologic choices may need tailoring to metabolic comorbidities.[14-18] Together, this literature argues that BMI and metabolic comorbidities are not merely background variables but active determinants of dermatologic phenotype and prognosis. Yet regional data from South Indian tertiary care linking the full spectrum of obesity-related skin changes to graded obesity and systematically profiled comorbidities remain limited, and local prevalence maps are essential to sharpen screening pathways and counselling.

Thus, the present study was conducted to document the spectrum of skin changes in obese individuals, assess their relationship with the severity of obesity, and explore possible associations with systemic comorbidities.

Materials and Methods

This cross-sectional observational study was conducted in the Department of Dermatology, Venereology and Leprosy, MediCiti Institute of Medical Sciences, Ghanpur (V), Medchal-Malkajgiri District, Telangana, over 18 months (January 2017-June 2018). Reporting adhered to STROBE guidance. IEC approvals obtained prior to this study.

Study Population: Adults (≥18 years) with obesity, defined for this study as BMI ≥30.0 kg/m², attending dermatology clinics during routine hours were screened and enrolled by consecutive sampling. Exclusion criteria were: refusal of consent; pregnancy or lactation; acute critical illness; primary endocrine disorders that independently alter skin phenotype (e.g., Cushing's syndrome); long-term systemic corticosteroid or other immunosuppressant use (>4 weeks within the prior 3 months); active malignancy; and known HIV infection. Obesity classes were assigned using WHO adult cut-offs: class I (30.0-34.9 kg/m²), class II (35.0-39.9 kg/m²), and class III (≥40.0 kg/m²). Sample size and characteristics. Ninety-five individuals met eligibility and were included (n=95). Of these, 70 were males (73.7%) and 25 were females (26.3%), yielding a male:female ratio of ~2.8:1. Most participants were 21-40 years old (60%).

Data collection procedures: A structured case-record form captured demographics, lifestyle factors (smoking, alcohol), symptom duration, prior dermatologic diagnoses/treatments, and medical history including type 2 diabetes mellitus, hypertension, dyslipidemia, hypothyroidism, and other cardiometabolic conditions. Current medications were recorded.

Anthropometry and blood pressure: Weight (to 0.1 kg) was measured on a calibrated digital scale with light clothing and no footwear; height (to 0.1 cm) on a wall-mounted stadiometer. BMI was computed as kg/m². Waist circumference was recorded at the midpoint between the lowest rib and iliac crest at end-expiration; hip circumference at the greater trochanters; waist-to-hip ratio (WHR) was derived. Blood pressure was measured with a validated automated sphygmomanometer after 5 minutes of seated rest; three readings were taken 1 minute apart, and the mean of the last two was used.

Dermatologic assessment: All participants underwent a head-to-toe examination by a board-certified dermatologist. A 10% random subset was re-evaluated by a senior dermatologist; any discrepancies were resolved by consensus. Diagnoses were clinical, supported where appropriate by point-of-care tests: 10-20% KOH mount for suspected dermatophytosis/candidiasis, Wood's lamp examination for pigmentary disorders, and dermoscopy for selected lesions. Validated grading/staging tools were used when applicable: Burke's scale for acanthosis nigricans; Hurley staging for hidradenitis suppurativa; lesion counts for acrochordons; Investigator Global Assessment or body-surface-area estimates for inflammatory dermatoses; and CEAP clinical class for chronic venous disease. The number of distinct dermatoses per participant was recorded.

Laboratory measurements and operational definitions: Fasting venous samples (8-12 h fast) were obtained the same day or within one week. Assays included fasting plasma glucose (FPG), HbA1c, and fasting lipid profile (total cholesterol, LDL-C, HDL-C, triglycerides). Thyroid-stimulating hormone was ordered when clinically indicated. Diabetes: known diabetes on therapy, FPG ≥ 126 mg/dL, or HbA1c ≥ 6.5%., Prediabetes: FPG 100-125 mg/dL or HbA1c 5.7-6.4%., Hypertension: known hypertension/on therapy or clinic BP ≥ 140/90 mmHg., Dyslipidemia: LDL-C ≥ 130 mg/dL, triglycerides ≥ 150 mg/dL, HDL-C < 40 mg/dL (men) or < 50 mg/dL (women), or lipid-lowering therapy. And Metabolic syndrome: IDF 2005 (South Asian cut-offs: waist ≥ 90 cm men/≥ 80 cm women) plus any two of: TG ≥ 150 mg/dL; low HDL-C (as above); BP ≥ 130/85 mmHg or on therapy; FPG ≥ 100 mg/dL or diabetes. Where available, non-alcoholic fatty liver disease was abstracted from contemporaneous ultrasound reports.

Targeted Outcomes of this study. Primary outcome: prevalence of specific cutaneous diagnoses across obesity classes. Secondary outcomes: (i) associations between BMI (continuous and class-based) and key dermatoses (e.g., acanthosis nigricans, acrochordons, intertrigo, superficial fungal infections, hidradenitis suppurativa, psoriasis, periorbital melanosis, plantar hyperkeratosis, venous stasis changes); (ii) associations between metabolic comorbidities (diabetes, hypertension, dyslipidemia, metabolic syndrome) and these dermatoses; and then (iii) count of distinct dermatoses per participant.

Statistical analysis: Analyses were performed using SPSS. Continuous variables are summarised as mean ± SD or median (IQR) as appropriate; categorical variables as counts and percentages. Group comparisons across obesity classes used

one-way ANOVA or Kruskal-Wallis tests (with post-hoc tests where indicated) and χ^2 tests (including χ^2 for trend) for categorical outcomes. Associations between BMI (per 5 kg/m² increment and by class) and common dermatoses were estimated as prevalence ratios via Poisson regression with robust variance; models were adjusted a priori for age and Gender, with sensitivity analyses adding diabetes, hypertension, and dyslipidemia. Correlations between adiposity indices (BMI, waist circumference, WHR) and lesion counts used Spearman's rho. Two-sided $p < 0.05$ meant statistical significance.

Results

Ninety-five adults with obesity were enrolled. Most were male (70/95, 73.7%), yielding a male: female ratio of ~2.8:1. Age skewed younger; 57/95 (60.0%) were 21-40 years. By BMI category, Class I predominated (79/95, 83.2%), with fewer in Class II (13/95, 13.7%) and Class III (3/95, 3.2%). Baseline distributions are presented in [Table 1].

Table 1: Baseline characteristics of the study cohort (n = 95)

Characteristic	n (%)
Gender	
Male	70 (73.7)
Female	25 (26.3)
Age (years)	
21-40	57 (60.0)
BMI class (WHO adult cut-offs)	
Class I (30.0-34.9 kg/m²)	79 (83.2)
Class II (35.0-39.9 kg/m²)	13 (13.7)
Class III (≥40.0 kg/m²)	3 (3.2)

Note: Values are n (%).

Figure 1: Acanthosis Nigricans & Acrochordons over the neck area

Figure 3: Straie Distensae

Figure 2: Giant Acrochordons over the face

Figure 4: Venous insufficiency with stasis eczema & ulcer

Figure 5: Xerosis

The cutaneous burden was broad [Table 2]. Acanthosis nigricans (AN) led the spectrum, affecting 62/95 (65.3%). Acrochordons were seen in 33/95 (34.7%) and striae distensae in 32/95 (33.7%). Other frequent findings included psoriasis (10/95, 10.5%), frictional hyperpigmentation (9/95, 9.5%), acne vulgaris (9/95, 9.5%), plantar hyperkeratosis (8/95, 8.4%), chronic venous insufficiency (8/95, 8.4%), and eczematous disorders (7/95, 7.4%). Pigmentary dermatoses melasma, photomelanosis, and lichen amyloidosis collectively accounted for 10/95 (10.5%). Representative images are shown in [Figure 1-6].

Figure 6: Chronic Eczema

Table 2: Spectrum of cutaneous diagnoses in obese adults.

Diagnosis	n (%)
Acanthosis nigricans	62 (65.3)
Acrochordons	33 (34.7)
Striae distensae	32 (33.7)
Psoriasis	10 (10.5)
Frictional hyperpigmentation	9 (9.5)
Acne vulgaris	9 (9.5)
Plantar hyperkeratosis	8 (8.4)
Chronic venous insufficiency (stasis changes)	8 (8.4)
Eczematous disorders	7 (7.4)
Pigmentary dermatoses (melasma, photomelanosis, lichen amyloidosis)	10 (10.5)

Note: Counts reflect diagnoses; individuals could have >1 condition. Values are n (%).

Across obesity classes, several dermatoses increased with higher BMI [Table 3]. AN occurred in 51/79 (64.6%) of Class I, 8/13 (61.5%) of Class II, and 3/3 (100%) of Class III. Frictional hyperpigmentation showed a similar gradient (3.8% in Class I; 23.1% in Class II; 100% in Class III). Plantar hyperkeratosis was uncommon in Class I (6.3%) and Class II (7.7%) but present in 2/3 (66.7%) in Class III. Psoriasis rose from 10.1% in Class I to 33.3% in Class III. These class-wise patterns suggest a BMI-linked intensification of metabolic and pressure-related skin changes; interpretations for Class III warrant caution given the small denominator (n = 3).

Table 3: Selected dermatoses by obesity class with trend testing

Diagnosis	Class I (n=79)	Class II (n=13)	Class III (n=3)	p for trend
Acanthosis nigricans	51/79 (64.6)	8/13 (61.5)	3/3 (100.0)	0.465
Frictional hyperpigmentation	3/79 (3.8)	3/13 (23.1)	3/3 (100.0)	<0.001
Plantar hyperkeratosis	5/79 (6.3)	1/13 (7.7)	2/3 (66.7)	0.008
Psoriasis	8/79 (10.1)	1/13 (7.7)	1/3 (33.3)	0.479

Note. Values are n/N within class (%). p for trend from the Cochran-Armitage test across Class I, then to III. Small Class III size (n= 3) inflates uncertainty; interpret p-values cautiously.

Infective dermatoses were documented in 41/95 (43.2%) [Table 4]. Dermatophytosis was most common (25/95, 26.3%). Intertrigo occurred in 5/95 (5.3%), scabies in 4/95 (4.2%), and pityriasis versicolor in 2/95 (2.1%). Less frequent infections included erythrasma, viral warts, herpes simplex/zoster, and trichomycosis axillaris (combined 5/95, 5.3%).

Table 4: Infective dermatoses in the cohort

Infection	n (%)
Dermatophytosis (tinea)	25 (26.3)
Intertrigo (bacterial/fungal)	5 (5.3)
Scabies	4 (4.2)
Pityriasis versicolor	2 (2.1)
Other (erythrasma, viral warts, HSV/HZ, trichomycosis)	5 (5.3)
Any infective dermatosis	41 (43.2)

Note: Values are n (%).

Metabolic comorbidities were prevalent [Table 5]. Hypertension was reported in 15/95 (15.7%) and diabetes mellitus in 12/95 (12.6%). Dyslipidaemia and polycystic ovarian disease were each present in 6/95 (6.3%), while hypothyroidism was noted in 5/95 (5.3%). In concert with the dermatosis profile especially AN, acrochordons, frictional hyperpigmentation, and plantar hyperkeratosis this comorbidity pattern underscores a tight clinicometabolic linkage within the cohort.

Table 5: Metabolic comorbidities (n = 95)

Comorbidity	n (%)
Hypertension	15 (15.7)
Diabetes mellitus	12 (12.6)
Dyslipidaemia	6 (6.3)
Polycystic ovarian disease	6 (6.3)
Hypothyroidism	5 (5.3)

Table Note: Values are n (%).

Discussion

Interpreting the Brief Findings: Obese adults in this South Indian clinic most often showed acanthosis nigricans (AN, 65.3%), acrochordons (34.7%), and striae (33.7%), with an appreciable infectious load (any infection 43.2%; dermatophytosis 26.3%). Several dermatoses rose with obesity class; frictional hyperpigmentation and plantar hyperkeratosis demonstrated dose-response trends ($p < 0.001$ and $p = 0.008$, respectively). Psoriasis was present in 10.5% and chronic venous insufficiency (CVI) in 8.4%. A small pigmentary cluster beyond AN (10.5%) was noted. Documented comorbidities were relatively modest (hypertension 15.7%, diabetes 12.6%, dyslipidemia 6.3%).

Comparison with existing literature: The pattern aligns with Indian clinic series in which AN and skin tags track closely with adiposity and insulin resistance.[2,3,6,7] Our infectious footprint mirrors reports from diabetes clinics where fungal disease, especially candidal and dermatophyte infections, concentrates in those with poorer glycemic control,[13] and agrees with case-control evidence that higher BMI independently associates with dermatophytosis.[8] The pigmentary signal beyond AN is consistent with periorbital melanosis linking to hyperinsulinemia, higher HOMA-IR, and an adverse leptin-adiponectin profile.[9] The CVI prevalence and its relationship with greater BMI echo vascular-surgery data showing higher CEAP stages and duplex-confirmed reflux with adiposity.[12] Our psoriasis share supports the well-described bidirectional psoriasis-metabolic axis obesity raises psoriasis risk and severity; metabolic burden can blunt responses to fixed-dose agents; and NAFLD and atherogenic lipids are common companions.[14–19] Mechanistically, the observed gradients fit with chronic low-grade inflammation (TNF-α/IL-23/IL-17), adipokine imbalance, mTORC1 signalling, and endothelial dysfunction as shared pathways across obesity-linked dermatoses.[1,3,4] Parallel Indian data reinforce broader cardio-cutaneous links HS with high metabolic-syndrome prevalence,[10] and pediatric AD with early metabolic risk,[11] contextualizing our adult findings within a wider continuum.

Deviations and plausible reasons: Two departures stand out. First, measured cardiometabolic comorbidities were lower than in some obesity or psoriasis cohorts that employed systematic biochemical screening.[7,15] Likely explanations include younger age distribution (60% aged 21-40), opportunistic rather than protocolized metabolic testing (no fasting insulin/HOMA-IR or adipokines), and under-ascertainment inherent to dermatology-led visits. Second, psoriasis trends by obesity class were directionally positive but statistically muted, plausibly due to small Class III numbers (n = 3) and limited power for trend testing at the severe-obesity tail. Notably, the high infection share despite modest known diabetes suggests contributions from pre-diagnostic insulin resistance and biomechanical/microenvironmental factors occlusion, friction, moisture, maceration that lower the threshold for fungal overgrowth.[1,2,13]

Study implications: Clinically, several bedside cues AN,

multiple tags, plantar hyperkeratosis, intertrigo, patterned hyperpigmentation should trigger a simple "screen-bundle": blood pressure, fasting glucose or HbA1c, fasting lipids, and waist circumference.[2,3,5] Routine case-finding for tinea/intertrigo with brief counseling on moisture control, garment fit, and early self-report is justified.[8,13] In psoriasis care, integrate weight management and select systemic therapy with metabolic profiles in mind; biologic choices and expectations of response may differ across BMI strata.[16–18] For CVI, quick ankle/CEAP screening and mobility/weight advice may reduce downstream morbidity.[12] At a service level, embedding protocolized metabolic phenotyping (including HOMA-IR where feasible) within dermatology clinics could surface occult risk earlier and align referrals to primary care/endocrinology.

Strengths and limitations: Strengths include standardised head-to-toe examination by specialists, use of point-of-care tests (KOH, Wood's lamp, dermoscopy) when indicated, and class-wise analyses that reveal biologically plausible gradients.

Limitations temper inference: cross-sectional design, single-center sampling with male predominance, very small Class III cell, inflating uncertainty at the highest BMI, and partial metabolic ascertainment without insulin resistance/adipokine panels.[7,9] Comorbidities were partly record-based, so dyslipidemia and NAFLD may be underestimated; phenotype severity metrics (e.g., PASI, uniform venous duplex) were not applied across all conditions. Future multi-center, prospective work with systematic metabolic profiling and longitudinal outcomes should test causality and quantify whether dermatology-led interventions improve both skin and cardiometabolic courses.

Conclusion

In this South Indian cohort, obesity aligned with a wide range of skin disorders that intensified with higher adiposity, alongside infectious, vascular, and inflammatory signals underscoring the skin as a visible marker of metabolic stress. Clinically, hallmark lesions related to insulin resistance, friction/pressure, or dysbiosis should cue a streamlined cardiometabolic screen and practical counselling (weight control, moisture management, proper garment/footwear fit); psoriasis care should integrate weight strategies, venous changes merit early attention, and protocolized metabolic phenotyping within dermatology can surface occult risk and enable coordinated co-management. Interpretation is limited by the single-centre, cross-sectional design and partial biochemical ascertainment. Future multicentre, prospective work with standardized severity metrics and comprehensive metabolic profiling should test causality and the effect of dermatology-led screening bundles; meanwhile, the cutaneous phenotype of obesity remains an actionable signal for targeted case-finding and coordinated care.

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Conflicts of interest

There are no conflicts of interest.

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