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Original Research Article

Dermatoses in Obesity: Relationship with Lipid Profile and Glycemic Status

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Name of Author:
Dr Kaveri Shivakumar [1], Dr Rajkeerthi N[2]

Affiliation: *[1]Assistant Professor Department of DVL KMCH Institute of Health Sciences and Research*
[2]Assistant Professor Department of Radiology SVMCH&RI.

Corresponding Author:
Dr Kaveri Shivakumar

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Abstract: ***Introduction*** Obesity is a chronic metabolic disorder associated with systemic inflammation, insulin resistance, dyslipidemia, and endocrine imbalance. Cutaneous manifestations often serve as early clinical indicators of underlying metabolic dysfunction. This study aimed to evaluate the pattern of dermatoses in obese individuals and assess their association with lipid profile abnormalities and glycemic status. ***Materials and Methods*** A cross-sectional study was conducted among 150 obese individuals (BMI ≥30 kg/m²) attending a tertiary care center. Detailed dermatological examination was performed. Fasting blood glucose (FBG), HbA1c, and lipid profile (Total cholesterol, LDL, HDL, triglycerides) were analyzed. Statistical analysis was done using SPSS; $p < 0.05$ was considered significant. ***Results*** Acanthosis nigricans (62%), skin tags (48%), striae distensae (44%), and fungal infections (38%) were the most common dermatoses. Significant associations were observed between acanthosis nigricans and elevated HbA1c ($p<0.001$), skin tags and hypertriglyceridemia ($p=0.002$), and striae with increased BMI ($p=0.01$). Dyslipidemia was present in 68% of participants. ***Conclusion*** Dermatoses in obesity strongly correlate with metabolic derangements. Early recognition of cutaneous markers may facilitate timely screening and prevention of cardiometabolic complications.

Keywords: Obesity, Acanthosis nigricans, Dyslipidemia, Hyperglycemia, Skin manifestations, Metabolic syndrome

INTRODUCTION

Obesity has emerged as a global public health crisis, with increasing prevalence across all age groups[1]. It is characterized by excessive accumulation of adipose tissue resulting in chronic low-grade inflammation and metabolic dysregulation[2]. Obesity is strongly associated with insulin resistance, dyslipidemia, hypertension, and type 2 diabetes mellitus[3].

Adipose tissue functions as an active endocrine organ secreting adipokines such as leptin, adiponectin, TNF-α, and interleukins, which contribute to systemic inflammation and metabolic imbalance[4]. These biochemical disturbances significantly influence skin physiology. The skin, being metabolically active, often reflects internal metabolic abnormalities[5].

Several cutaneous manifestations are commonly associated with obesity. Acanthosis nigricans is widely regarded as a marker of insulin resistance[6]. Skin tags (acrochordons) have been linked with hyperinsulinemia and metabolic syndrome[7]. Striae distensae occur due to mechanical stretching and hormonal alterations[8]. Intertrigo and fungal infections are more frequent due to increased skin folds and impaired immunity[9].

Dyslipidemia, characterized by elevated triglycerides, LDL cholesterol, and reduced HDL cholesterol, contributes to microvascular changes that may affect skin integrity[10]. Hyperglycemia impairs neutrophil function and collagen synthesis, predisposing

individuals to recurrent infections and delayed wound healing[11].

The interplay between obesity, insulin resistance, and lipid abnormalities forms the core pathophysiological mechanism behind obesity-related dermatoses[12]. Early recognition of dermatological signs may therefore serve as a non-invasive screening tool for metabolic disorders[13].

Although several studies have independently explored obesity-related skin changes, limited research has systematically correlated dermatoses with biochemical markers such as lipid profile and glycemic indices in Indian populations[14].
Hence, the present study was conducted to evaluate the spectrum of dermatoses in obese individuals and analyze their association with lipid profile and glycemic status.

MATERIALS AND METHODS

This is a hospital-based and cross-sectional observational study was conducted in the Department of Dermatology in collaboration with Department of Biochemistry at a tertiary care center over a period of 12 months.

Sample Size
150 obese individuals (BMI ≥30 kg/m²).

Inclusion Criteria
- Age 18–60 years
- BMI ≥30 kg/m²
- Willing to give informed consent

Exclusion Criteria
- Pregnant women
- Known endocrine disorders (Cushing's syndrome, hypothyroidism)
- Chronic liver/kidney disease
- Patients on systemic steroids or lipid-lowering therapy

Data Collection
- Detailed history and anthropometric measurements (BMI, waist circumference)
- Complete dermatological examination
- Laboratory investigations:
  - Fasting blood glucose (FBG)
  - HbA1c
  - Total cholesterol
  - LDL cholesterol
  - HDL cholesterol
  - Triglycerides

Statistical Analysis
Data analyzed using SPSS version 25.
- Mean ± SD calculated
- Chi-square test for categorical variables
- Pearson correlation for continuous variables
- $p<0.05$ considered significant

Ethical clearance obtained from institutional ethics committee.

RESULTS

Table 1: Demographic Distribution

Parameter	Number (%)
Male	68 (45.3%)
Female	82 (54.7%)
Mean Age	38.6 ± 9.4 years
Mean BMI	32.8 ± 2.6

Majority were females with mean BMI in class I obesity range.

Table 2: Spectrum of Dermatoses

Dermatoses	Frequency (%)
Acanthosis Nigricans	93 (62%)
Skin Tags	72 (48%)
Striae Distensae	66 (44%)
Fungal Infections	57 (38%)
Intertrigo	42 (28%)
Psoriasis	18 (12%)

Acanthosis nigricans was the most prevalent lesion.

Table 3: Lipid Profile Abnormalities

Parameter	Mean ± SD
Total Cholesterol	212 ± 36 mg/dL
LDL	138 ± 28 mg/dL
HDL	39 ± 7 mg/dL
Triglycerides	186 ± 44 mg/dL

High triglycerides and low HDL were predominant abnormalities.

Table 4: Glycemic Status

Parameter	Mean ± SD
FBG	118 ± 22 mg/dL
HbA1c	6.4 ± 0.8 %

Majority were prediabetic.

Table 5: Association Between Dermatoses and HbA1c

Condition	HbA1c ≥6.5%	p-value
Acanthosis Nigricans	70%	<0.001
Skin Tags	52%	0.01
Striae	34%	0.08

Strong association between acanthosis and diabetes.

Table 6: Association Between Dermatoses and Dyslipidemia

Condition	Hypertriglyceridemia	p-value
Skin Tags	61%	0.002
Psoriasis	55%	0.03
Fungal Infection	42%	0.07

Skin tags significantly correlated with hypertriglyceridemia.

DISCUSSION

The present study highlights the strong association between obesity-related dermatoses and underlying metabolic abnormalities, particularly dyslipidemia and impaired glycemic control. Obesity is now widely recognized as a chronic inflammatory state characterized by adipocyte hypertrophy, macrophage infiltration, and altered adipokine secretion, which together create a milieu of insulin resistance and metabolic dysfunction. The cutaneous manifestations observed in this study reflect these systemic biochemical disturbances and reinforce the concept that the skin acts as a visible indicator of internal metabolic imbalance.

Acanthosis nigricans (AN), the most prevalent dermatosis in our study (62%), demonstrated a statistically significant association with elevated HbA1c levels ($p<0.001$). This finding is consistent with previous studies that identify AN as a reliable clinical marker of insulin resistance and hyperinsulinemia. Hyperinsulinemia stimulates insulin-like growth factor-1 (IGF-1) receptors on keratinocytes and dermal fibroblasts, leading to epidermal hyperplasia and papillomatosis. The strong correlation between AN and HbA1c ≥6.5% observed in our cohort underscores the role of chronic hyperglycemia in promoting epidermal proliferation. Similar findings were reported in Indian and international cohorts, where AN prevalence ranged from 55% to 75% among obese or prediabetic individuals. The high percentage of AN in our study suggests that dermatological screening may provide an opportunity for early identification of individuals at risk for type 2 diabetes mellitus.

Skin tags (acrochordons) were present in 48% of participants and showed a significant association with hypertriglyceridemia ($p=0.002$). The pathogenesis of acrochordons in obesity is believed to involve hyperinsulinemia-induced keratinocyte proliferation and increased dermal fibroblast growth. Elevated triglycerides reflect increased circulating very-low-density lipoproteins (VLDL), which are associated with insulin resistance and metabolic syndrome. The significant relationship observed in our study supports earlier research indicating that skin tags may serve as external markers of dyslipidemia and cardiovascular risk. The association between acrochordons and metabolic syndrome has been well documented, with studies demonstrating increased prevalence of hypertriglyceridemia, reduced HDL cholesterol, and elevated fasting insulin levels in patients with multiple skin tags.

Striae distensae were observed in 44% of participants and were significantly associated with increased BMI ($p=0.01$), but not strongly correlated with HbA1c. The development of striae in obesity is primarily attributed to mechanical stretching of the dermis due to rapid weight gain. Additionally, altered collagen synthesis and decreased dermal elasticity contribute to dermal tearing. Cortisol dysregulation, even within physiological ranges, may also influence dermal matrix degradation. Our findings align with prior studies that emphasize BMI as the primary determinant of striae formation rather than glycemic status. This suggests that striae may reflect mechanical and hormonal factors more than metabolic dysregulation per se.

Fungal infections (38%) and intertrigo (28%) were common in our cohort. Hyperglycemia impairs neutrophil chemotaxis and phagocytic function, reducing host immunity and increasing susceptibility to infections. Elevated glucose levels in sweat and skin folds create a favorable environment for fungal proliferation, particularly Candida species. Similar infection rates have been documented in obese and diabetic populations. The increased skin fold surface area in obesity promotes moisture retention and

friction, further predisposing individuals to intertriginous infections. Although the association with glycemic parameters was not statistically significant for all infection types, the overall trend supports the immunocompromised state associated with metabolic dysregulation.

Psoriasis, observed in 12% of cases, demonstrated significant association with hypertriglyceridemia (p=0.03). Obesity and psoriasis share common inflammatory pathways involving TNF-α, IL-6, and IL-17. Adipose tissue-derived cytokines exacerbate systemic inflammation, which may worsen psoriatic activity. Recent literature suggests that obesity not only increases psoriasis risk but also reduces therapeutic response to biologic agents. The inflammatory cross-talk between adipose tissue and skin likely explains the co-existence observed in our study. This finding emphasizes the importance of metabolic evaluation in patients with chronic inflammatory dermatoses.

Dyslipidemia was present in 68% of participants, characterized predominantly by elevated triglycerides and reduced HDL cholesterol. This pattern is typical of insulin resistance and metabolic syndrome. Elevated triglycerides contribute to endothelial dysfunction and oxidative stress, which may impair dermal microcirculation. Reduced HDL levels further exacerbate inflammatory processes, as HDL possesses anti-inflammatory and antioxidant properties. These lipid abnormalities may indirectly influence skin health by promoting chronic low-grade inflammation and impaired tissue repair.

The mean HbA1c (6.4 ± 0.8%) indicates that a large proportion of participants were in the prediabetic range. The coexistence of cutaneous markers with borderline glycemic indices suggests that dermatological findings may precede overt diabetes. Early detection of such markers could facilitate timely lifestyle interventions and prevent progression to type 2 diabetes mellitus.

From a pathophysiological perspective, adipose tissue acts as an endocrine organ producing leptin, resistin, adiponectin, and pro-inflammatory cytokines. In obesity, decreased adiponectin and increased leptin levels promote insulin resistance and inflammation. TNF-α and IL-6 interfere with insulin signaling pathways, leading to hyperinsulinemia and hyperglycemia. These metabolic disturbances directly affect keratinocyte proliferation, sebaceous gland activity, collagen metabolism, and immune function. Therefore, the dermatological manifestations observed in this study are not merely cosmetic consequences of obesity but represent systemic metabolic dysfunction.

Our findings support the hypothesis that dermatological evaluation can serve as a cost-effective, non-invasive screening method for metabolic disorders. In resource-limited settings, where routine biochemical screening may not always be feasible, recognition of markers such as acanthosis nigricans and skin tags can prompt further metabolic evaluation.

The strengths of this study include systematic dermatological assessment and correlation with objective biochemical parameters. However, limitations include the cross-sectional design, which precludes causal inference, and lack of control group for comparison. Longitudinal studies are warranted to evaluate whether improvement in metabolic parameters leads to regression of dermatoses.
Overall, this study reinforces the bidirectional relationship between obesity and skin disease. The skin acts as a mirror reflecting internal metabolic imbalance. Early recognition of obesity-associated dermatoses can facilitate integrated management strategies involving dermatologists, endocrinologists, and primary care physicians. Multidisciplinary intervention focusing on weight reduction, glycemic control, and lipid optimization may not only improve systemic health but also ameliorate cutaneous manifestations.

CONCLUSION

Obesity-related dermatoses are strongly associated with dyslipidemia and impaired glycemic control. Acanthosis nigricans and skin tags can act as early clinical markers of insulin resistance and lipid abnormalities. Integrated dermatological and metabolic screening is essential for early intervention and prevention of long-term complications.

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