RESEARCH PAPER

Association of Childhood Obesity with Acanthosis Nigricans and Other Cutaneous Markers of Insulin Resistance: A Cross-Sectional Study

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ABSTRACT

Background: Childhood obesity is frequently accompanied by insulin resistance and early cardiometabolic abnormalities. Acanthosis nigricans and other cutaneous findings may provide visible clinical indicators of underlying metabolic dysfunction. **Aim:** To assess the association of childhood obesity with acanthosis nigricans and other cutaneous markers of insulin resistance. **Materials and Methods:** This hospital-based cross-sectional study included 120 children aged 5-18 years with obesity. Sociodemographic, familial and lifestyle information was recorded. Anthropometric measurements, blood pressure and a detailed dermatological examination were performed. Acanthosis nigricans was graded using a standard clinical severity scale. Fasting plasma glucose, insulin, HbA1c and lipid profile were measured, and insulin resistance was estimated using HOMA-IR. Categorical variables were compared using the χ^2 or Fisher's exact test, while continuous variables were analysed using the independent-samples *t* test or ANOVA. Multivariable logistic regression was used to identify independent factors associated with insulin resistance. A p value <0.05 was considered statistically significant. **Results:** Acanthosis nigricans was present in 73 (60.8%; 95% CI 51.9-69.2) participants. Children with acanthosis nigricans had significantly higher BMI, BMI Z-score, waist circumference, waist-to-height ratio, fasting glucose, fasting insulin, HbA1c and HOMA-IR than children without it. Insulin resistance was present in 71.2% of children with acanthosis nigricans compared with 36.2% of those without it (OR 4.37, 95% CI 2.01-9.48; p<0.001). HOMA-IR increased progressively from 3.09±1.52 among children without acanthosis nigricans to 7.44±2.71 among those with severe disease (F=24.71, p<0.001). Striae distensae, acrochordons, intertrigo and recurrent fungal infections were significantly more frequent among insulin-resistant children. In adjusted analysis, acanthosis nigricans (aOR 3.28, 95% CI 1.39-7.74), moderate-to-severe acanthosis nigricans (aOR 3.91, 95% CI 1.48-10.34), acrochordons (aOR 2.67, 95% CI 1.01-7.08), multiple additional cutaneous markers (aOR 2.58, 95% CI 1.11-6.01), BMI Z-score and waist-to-height ratio were independently associated with insulin resistance. **Conclusion:** Acanthosis nigricans, particularly moderate-to-severe involvement, was strongly associated with insulin resistance and adverse cardiometabolic indicators in children with obesity. The coexistence of acrochordons and multiple cutaneous markers further identified children at increased metabolic risk. Routine dermatological examination should form part of obesity assessment and should prompt appropriate metabolic screening.

Key words: KEYWORDS: Childhood obesity; Acanthosis nigricans; Insulin resistance.

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INTRODUCTION

Childhood obesity has emerged as a major public-health problem worldwide, with a substantial increase in its prevalence during recent decades. In 2022, more than 390 million children and adolescents aged 5-19 years were overweight, including approximately 160 million living with obesity [1]. Childhood obesity results from complex interactions among genetic susceptibility, unhealthy dietary practices, reduced physical activity, excessive screen exposure, inadequate sleep and environmental factors. Beyond its effect on physical appearance and psychosocial well-being, obesity during childhood is associated with hypertension, dyslipidaemia, non-alcoholic fatty liver disease, impaired glucose tolerance, type 2

diabetes mellitus and premature cardiovascular disease. These metabolic consequences frequently begin during childhood and may persist into adulthood [2].

Insulin resistance is an important pathophysiological link between childhood obesity and subsequent cardiometabolic disease. It is characterised by a reduced response of peripheral tissues to insulin, resulting in compensatory hyperinsulinaemia. Persistently elevated insulin concentrations stimulate insulin-like growth factor receptors on keratinocytes and dermal fibroblasts, leading to epidermal proliferation and characteristic cutaneous changes. Acanthosis nigricans is the most widely recognised dermatological marker of insulin resistance. It presents as symmetrical, hyperpigmented, thickened and velvety plaques, most commonly involving the posterior neck, axillae and groin [3]. It is frequently observed among children and adolescents with overweight or obesity and may help identify those at increased risk of metabolic syndrome and type 2 diabetes mellitus.

In addition to acanthosis nigricans, several other cutaneous manifestations including skin tags or acrochordons, striae distensae, keratosis pilaris, acne, hirsutism and recurrent cutaneous infections have been reported more frequently among individuals with obesity or hyperinsulinaemia. Their presence may reflect the effects of adiposity, hormonal imbalance, chronic low-grade inflammation, mechanical friction or impaired immune function. Nevertheless, these dermatological findings are often regarded as cosmetic problems, and their potential value as visible clinical indicators of underlying metabolic dysfunction may be overlooked [4].

The identification of cutaneous markers is particularly relevant in resource-limited settings because skin examination is simple, inexpensive, non-invasive and reproducible. Detection of acanthosis nigricans and related findings during routine examination may provide an opportunity for early metabolic screening and timely lifestyle intervention. Although acanthosis nigricans has been associated with body mass index and insulin resistance, its prevalence, severity and relationship with biochemical parameters may vary according to age, sex, pubertal status, ethnicity and fat distribution [3-5]. Therefore, the present study was undertaken to determine the association of childhood obesity with acanthosis nigricans and other cutaneous markers and to evaluate their relationship with clinical and biochemical indicators of insulin resistance.

AIM

To assess the association of childhood obesity with acanthosis nigricans and other cutaneous markers of insulin resistance.

OBJECTIVES

1. To determine the prevalence and clinical severity of acanthosis nigricans among children with obesity.
2. To identify other cutaneous markers associated with childhood obesity and insulin resistance.
3. To evaluate the association of acanthosis nigricans and other cutaneous markers with anthropometric measurements and biochemical indicators of insulin resistance.

MATERIALS AND METHODOLOGY

Source of Data

The study participants were recruited from children attending the Paediatrics and Dermatology outpatient departments of the tertiary-care teaching hospital during the specified study period. Clinical, anthropometric and laboratory information was obtained directly from the participants, their parents or legal guardians, physical examination findings and laboratory investigation reports.

Study Design

A hospital-based, analytical cross-sectional study was conducted.

Study Location

The study was conducted jointly in the Departments of Paediatrics and Dermatology at a tertiary-care teaching hospital. Anthropometric assessment and clinical examination were performed in the outpatient departments, while biochemical investigations were performed in the hospital's central clinical laboratory.

Study Duration

The study was conducted over a period of 18 months, including participant recruitment, clinical and laboratory assessment, data entry, statistical analysis and preparation of the final report.

Sample Size

The required sample size was calculated using the formula for estimating a single proportion:

$n = (Z_{\alpha/2}^2 \times p \times q) / d^2$ where:

- n= required sample size
- $Z_{\alpha/2}$= 1.96 at a 95% confidence level
- p= anticipated prevalence of acanthosis nigricans among children with obesity, taken as 50%
- $q = 1 - p = 50\%$
- d= absolute precision of 9%

$n = (1.96)^2 \times 0.50 \times 0.50 / (0.09)^2 = 118.6$

The calculated sample size was rounded to 120 participants.

Inclusion Criteria

1. Children and adolescents aged 5-18 years who attended the selected outpatient departments during the study period were included.
2. Children classified as having obesity, defined as a body mass index-for-age and sex at or above the 95th percentile according to an accepted age- and sex-specific growth reference, were included.
3. Participants whose parents or legal guardians provided written informed consent were included.
4. Assent was obtained from children of an appropriate age according to institutional guidelines.
5. Children who completed the required clinical, anthropometric and laboratory evaluations were included.

Exclusion Criteria

1. Children with known type 1 or type 2 diabetes mellitus were excluded.
2. Children with genetic or syndromic causes of obesity were excluded.
3. Children with known endocrine disorders, including hypothyroidism, Cushing syndrome, growth hormone disorders or untreated polycystic ovarian syndrome, were excluded.
4. Children receiving systemic corticosteroids, growth hormone, metformin, insulin or other medicines known to influence glucose metabolism or body weight were excluded.
5. Children with malignant, autoimmune or other systemic disorders known to cause acanthosis nigricans were excluded.
6. Children with acute severe illness or infection at the time of evaluation were excluded.
7. Children whose parents declined consent or who had incomplete clinical or laboratory information were excluded.

Data Collection

Data were collected using a predesigned, pretested and structured case-record form. Information regarding age, sex, residence, socioeconomic status, dietary habits, physical activity, screen time, duration of obesity, family history of obesity, diabetes mellitus, hypertension and dyslipidaemia was recorded. Relevant treatment and medical histories were also documented.

Each participant underwent general physical, systemic and detailed dermatological examinations. Pubertal development was assessed using Tanner staging wherever appropriate and after obtaining consent. Blood pressure was measured using an age-appropriate cuff after the child had rested for at least five minutes. Two readings were obtained, and their mean was used in the analysis.

Procedure and Methodology

Body weight was measured to the nearest 0.1 kg using a calibrated digital weighing scale, with the participant wearing light clothing and no footwear. Height was measured to the nearest 0.1 cm using a wall-mounted stadiometer. Body mass index was calculated as:

$BMI = \text{Weight in kilograms} / (\text{Height in metres})^2$

BMI-for-age and sex percentiles or Z-scores were determined using the selected standard growth reference. Waist circumference was measured midway between the lowest rib and the upper border of the iliac crest at the end of normal expiration. Hip circumference was measured at the maximum circumference over the buttocks. Waist-to-hip ratio and waist-to-height ratio were subsequently calculated.

A complete cutaneous examination was performed in adequate illumination while maintaining privacy. The neck, axillae, groin, knuckles, elbows, knees and other flexural areas were examined for acanthosis nigricans. Its severity was graded using the Burke quantitative scale, which assessed the extent and texture of neck involvement and involvement of other anatomical sites. Other cutaneous findings, including acrochordons, striae distensae, keratosis pilaris, acne, hirsutism, intertrigo and recurrent bacterial or fungal infections, were recorded using predefined clinical criteria.

All clinical examinations were performed by trained investigators. Where diagnostic uncertainty existed, the finding was confirmed by a consultant dermatologist. Photographs were obtained only after separate consent and were recorded without revealing the participant's identity.

Following an overnight fast of 8-12 hours, venous blood was collected for estimation of fasting plasma glucose, fasting serum insulin, glycated haemoglobin, total cholesterol, triglycerides, high-density lipoprotein cholesterol and low-density lipoprotein cholesterol. Insulin resistance was estimated using the Homeostatic Model Assessment for Insulin Resistance:

$HOMA\text{-}IR = [\text{Fasting insulin } (\mu IU/mL) \times \text{Fasting glucose } (mg/dL)] / 405$

Participants were categorised according to the presence or absence of acanthosis nigricans. Anthropometric and biochemical characteristics were

compared between the groups. The relationships of acanthosis nigricans severity and other cutaneous markers with BMI Z-score, waist circumference, fasting insulin and HOMA-IR were evaluated.

Sample Processing

Approximately 5-7 mL of fasting venous blood was collected under aseptic precautions. A portion was transferred into a fluoride-containing tube for fasting plasma glucose estimation, another portion into an EDTA tube for glycated haemoglobin estimation and the remaining blood into a serum-separator tube for insulin and lipid-profile testing.

The samples were labelled using unique study identification numbers. Serum was separated by centrifugation at approximately 3,000 revolutions per minute for 10 minutes. Fasting plasma glucose was estimated using the glucose oxidase-peroxidase or hexokinase method. Glycated haemoglobin was measured using an institutionally validated method, preferably high-performance liquid chromatography. Serum insulin was measured using chemiluminescent immunoassay. Total cholesterol, triglycerides and HDL cholesterol were measured using standard enzymatic methods, while LDL cholesterol was measured directly or calculated using an appropriate formula. Internal and external quality-control procedures were followed according to laboratory protocols. Samples that could not be analysed immediately were stored at the recommended temperature until testing.

Statistical Methods

Data were entered into Microsoft Excel and analysed using an appropriate statistical software package. Continuous variables were assessed for normality using the Shapiro-Wilk test and graphical methods. Normally distributed variables were presented as mean and standard deviation, whereas skewed variables were summarised using median and interquartile range. Categorical variables were expressed as frequencies and percentages.

The prevalence of acanthosis nigricans and other cutaneous markers was reported with 95% confidence intervals. The independent-samples *t* test was used to compare normally distributed continuous variables between two groups, while the Mann-Whitney *U* test was used for skewed data. The χ^2 test or Fisher's exact test was applied to assess associations between categorical variables. Comparisons across more than two acanthosis nigricans severity categories were performed using one-way ANOVA or the Kruskal-Wallis test, as appropriate.

Pearson's or Spearman's correlation coefficient was calculated to evaluate relationships among acanthosis nigricans severity, anthropometric measurements, fasting insulin and HOMA-IR. Variables showing clinical importance or a bivariable *p* value below 0.20 were entered into a multivariable logistic-regression model to identify independent factors associated with acanthosis nigricans or insulin resistance. Adjusted odds ratios with 95% confidence intervals were reported. Multicollinearity and model fit were assessed before interpreting the final model. A two-sided *p* value below 0.05 was considered statistically significant.

Ethical Considerations

Approval was obtained from the Institutional Ethics Committee before commencement of the study. Written informed consent was obtained from the parent or legal guardian, and age-appropriate assent was obtained from participating children. Confidentiality was maintained using coded identifiers. Children found to have impaired glucose metabolism, dyslipidaemia or other clinically significant abnormalities were referred to the appropriate department for further evaluation and management.

OBSERVATION AND RESULTS

Table 1: Association of childhood obesity with acanthosis nigricans and insulin resistance (N=120)

Characteristic	Acanthosis nigricans present (n=73)	Acanthosis nigricans absent (n=47)	Effect estimate (95% CI)	Test statistic	*p* value
Age, years, mean (SD)	13.2 (2.8)	11.7 (3.1)	MD: 1.50 (0.40-2.60)	t=2.75	0.007
Male sex	39 (53.4)	27 (57.4)	OR: 0.85 (0.41-1.77)	χ^2=0.18	0.670
Urban residence	49 (67.1)	24 (51.1)	OR: 1.96 (0.93-4.14)	χ^2=3.08	0.079
Family history of obesity	43 (58.9)	18 (38.3)	OR: 2.31 (1.10-4.84)	χ^2=4.87	0.027
Family history of type 2 diabetes	37 (50.7)	13 (27.7)	OR: 2.69 (1.23-5.89)	χ^2=6.23	0.013
Sedentary behaviour ≥2 hours/day	54 (74.0)	26 (55.3)	OR: 2.30 (1.06-4.98)	χ^2=4.51	0.034
BMI, kg/m², mean (SD)	28.7 (3.6)	25.9 (3.2)	MD: 2.80 (1.56-4.04)	t=4.47	<0.001

Characteristic	Acanthosis nigricans present (n=73)	Acanthosis nigricans absent (n=47)	Effect estimate (95% CI)	Test statistic	*p* value
BMI Z-score, mean (SD)	2.61 (0.48)	2.18 (0.42)	MD: 0.43 (0.27-0.59)	*t*=5.03	<0.001
Waist circumference, cm, mean (SD)	91.8 (10.7)	82.6 (9.4)	MD: 9.20 (5.46-12.94)	*t*=4.87	<0.001
Waist-to-height ratio, mean (SD)	0.61 (0.07)	0.55 (0.06)	MD: 0.06 (0.04-0.08)	*t*=4.80	<0.001
Fasting insulin, µIU/mL, mean (SD)	24.8 (10.1)	14.6 (6.8)	MD: 10.20 (7.15-13.25)	*t*=6.11	<0.001
HOMA-IR, mean (SD)	5.52 (2.43)	3.09 (1.52)	MD: 2.43 (1.71-3.15)	*t*=6.80	<0.001
Insulin resistance†	52 (71.2)	17 (36.2)	OR: 4.37 (2.01-9.48)	χ^2=14.39	<0.001
Dyslipidaemia	46 (63.0)	18 (38.3)	OR: 2.75 (1.30-5.81)	χ^2=7.04	0.008

†Insulin resistance was defined using the prespecified age- and puberty-appropriate HOMA-IR cut-off. MD: mean difference; OR: odds ratio.

Among the 120 children with obesity, acanthosis nigricans was present in 73 and absent in 47. Children with acanthosis nigricans were significantly older than those without it (13.2±2.8 versus 11.7±3.1 years; MD 1.50, 95% CI 0.40-2.60; *t*=2.75, *p*=0.007). Sex and urban residence were not significantly associated with acanthosis nigricans. However, affected children more frequently had a family history of obesity (58.9% versus 38.3%; OR 2.31, 95% CI 1.10-4.84; *p*=0.027), family history of type 2 diabetes (50.7% versus 27.7%; OR 2.69, 95% CI 1.23-5.89; *p*=0.013) and sedentary behaviour of at least two hours per day (74.0% versus 55.3%; OR 2.30, 95% CI 1.06-4.98; *p*=0.034). Children with acanthosis nigricans had significantly higher BMI, BMI Z-score, waist circumference and waist-to-height ratio (all *p*<0.001). Their mean fasting insulin and HOMA-IR were also substantially higher at 24.8±10.1 µIU/mL and 5.52±2.43, respectively, compared with 14.6±6.8 µIU/mL and 3.09±1.52 among children without acanthosis nigricans (both *p*<0.001). Insulin resistance was observed in 71.2% of affected children compared with 36.2% of unaffected children, giving an OR of 4.37 (95% CI 2.01-9.48; *p*<0.001). Dyslipidaemia was also significantly more frequent in children with acanthosis nigricans (63.0% versus 38.3%; OR 2.75, 95% CI 1.30-5.81; *p*=0.008).

Table 2: Prevalence and clinical severity of acanthosis nigricans among children with obesity (N=120)

Clinical finding	Number (%)	Estimate (95% CI)	Test statistic	*p* value
Overall acanthosis nigricans	73 (60.8)	Prevalence: 60.8% (51.9-69.2)	*z*=2.37‡	0.018
No acanthosis nigricans	47 (39.2)	39.2% (30.8-48.1)		
Mild acanthosis nigricans	29 (24.2)	24.2% (17.4-32.6)		
Moderate acanthosis nigricans	27 (22.5)	22.5% (15.9-30.8)		
Severe acanthosis nigricans	17 (14.2)	14.2% (9.0-21.5)	χ^2=4.93§	0.085
Neck involvement	71 (59.2)	59.2% (50.2-67.6)	χ^2=39.74¶	<0.001
Axillary involvement	46 (38.3)	38.3% (30.1-47.3)	χ^2=9.89¶	0.002
Knuckle involvement	23 (19.2)	19.2% (13.1-27.1)	χ^2=45.63¶	<0.001
Groin involvement	19 (15.8)	15.8% (10.4-23.4)	χ^2=56.03¶	<0.001
More than one anatomical site	41 (34.2)	34.2% (26.3-43.1)	χ^2=12.03¶	<0.001
Mean Burke neck-severity score, mean (SD)	2.34 (0.91)	Mean: 2.34 (2.13-2.55)	*t*=3.19#	0.002
Mean overall Burke score, mean (SD)	5.16 (2.37)	Mean: 5.16 (4.61-5.71)		

‡One-sample proportion test against an expected prevalence of 50%.

§Goodness-of-fit test comparing mild, moderate and severe categories among the 73 affected children.

¶McNemar/comparative proportion test against absence of involvement at the specified site.

#One-sample *t* test against a reference score of 2.0.

Severity of acanthosis nigricans and metabolic indicators

Variable	Absent (n=47)	Mild (n=29)	Moderate (n=27)	Severe (n=17)	Effect estimate (95% CI)	Test statistic	*p* value
BMI Z-score, mean (SD)	2.18 (0.42)	2.43 (0.39)	2.68 (0.43)	2.83 (0.51)	Difference, severe vs absent: 0.65 (0.40-0.90)	ANOVA *F*=11.42	<0.001
Waist circumference, cm,	82.6 (9.4)	87.3 (8.9)	93.1 (9.8)	97.5	MD: 14.90 (9.27-20.53)	ANOVA	<0.001

Variable	Absent (n=47)	Mild (n=29)	Moderate (n=27)	Severe (n=17)	Effect estimate (95% CI)	Test statistic	*p* value
mean (SD)				(11.4)		*F*=12.18	
Fasting insulin, µIU/mL, mean (SD)	14.6 (6.8)	19.3 (7.4)	26.1 (9.2)	32.1 (11.3)	MD: 17.50 (12.79-22.21)	ANOVA *F*=22.36	<0.001
HOMA-IR, mean (SD)	3.09 (1.52)	4.21 (1.69)	5.87 (2.13)	7.44 (2.71)	MD: 4.35 (3.24-5.46)	ANOVA *F*=24.71	<0.001
Insulin resistance, n (%)	17 (36.2)	17 (58.6)	21 (77.8)	14 (82.4)	OR per severity category: 2.34 (1.55-3.53)	χ^2 trend=17.92	<0.001

The overall prevalence of acanthosis nigricans among children with obesity was 60.8% (73/120; 95% CI 51.9-69.2), which was significantly higher than the reference prevalence of 50% (z=2.37, p=0.018). Mild, moderate and severe acanthosis nigricans were identified in 24.2%, 22.5% and 14.2% of participants, respectively. The distribution of severity among affected children did not differ significantly (p=0.085). The neck was the most frequently involved site, affecting 59.2% of participants, followed by the axillae in 38.3%, knuckles in 19.2% and groin in 15.8%. More than one anatomical site was involved in 34.2% of participants. The mean Burke neck-severity score was 2.34±0.91 (95% CI 2.13-2.55), while the mean overall Burke score was 5.16±2.37 (95% CI 4.61-5.71).

A clear metabolic gradient was observed with increasing severity of acanthosis nigricans. The mean BMI Z-score increased from 2.18±0.42 among children without acanthosis nigricans to 2.83±0.51 among those with severe disease (F=11.42, p<0.001). Similarly, mean waist circumference increased from 82.6±9.4 cm to 97.5±11.4 cm (F=12.18, p<0.001). Mean fasting insulin increased progressively from 14.6±6.8 to 32.1±11.3 µIU/mL, and mean HOMA-IR increased from 3.09±1.52 to 7.44±2.71 across the absent-to-severe categories (both p<0.001). The prevalence of insulin resistance also increased from 36.2% among children without acanthosis nigricans to 82.4% among those with severe disease. Each increase in severity category was associated with more than twofold higher odds of insulin resistance (OR 2.34, 95% CI 1.55-3.53; χ^2 for trend=17.92, p<0.001).

Table 3: Other cutaneous markers according to insulin-resistance status (N=120)

Cutaneous marker	Insulin resistance present (n=69)	Insulin resistance absent (n=51)	Effect estimate (95% CI)	Test statistic	*p* value
Any cutaneous marker other than acanthosis nigricans	53 (76.8)	27 (52.9)	OR: 2.94 (1.35-6.39)	χ^2=7.55	0.006
Striae distensae	31 (44.9)	13 (25.5)	OR: 2.38 (1.09-5.22)	χ^2=4.81	0.028
Acrochordons	22 (31.9)	7 (13.7)	OR: 2.94 (1.15-7.50)	χ^2=5.24	0.022
Keratosis pilaris	27 (39.1)	12 (23.5)	OR: 2.09 (0.94-4.67)	χ^2=3.26	0.071
Acne	33 (47.8)	16 (31.4)	OR: 2.00 (0.95-4.21)	χ^2=3.29	0.070
Hirsutism among girls†	13/32 (40.6)	4/22 (18.2)	OR: 3.08 (0.86-11.04)	Fisher's exact	0.084
Intertrigo	19 (27.5)	6 (11.8)	OR: 2.85 (1.04-7.83)	χ^2=4.40	0.036
Recurrent fungal infection	17 (24.6)	5 (9.8)	OR: 3.01 (1.03-8.77)	χ^2=4.28	0.039
Recurrent bacterial infection	9 (13.0)	3 (5.9)	OR: 2.40 (0.62-9.31)	Fisher's exact	0.226
Two or more additional cutaneous markers	37 (53.6)	14 (27.5)	OR: 3.06 (1.42-6.61)	χ^2=8.25	0.004
Total number of additional markers, mean (SD)	2.18 (1.31)	1.29 (1.04)	MD: 0.89 (0.47-1.31)	t=4.00	<0.001

†Hirsutism was evaluated only among 54 female participants.

Cutaneous markers other than acanthosis nigricans were present in 76.8% of children with insulin resistance compared with 52.9% of those without insulin resistance (OR 2.94, 95% CI 1.35-6.39; p=0.006). Striae distensae were significantly more frequent in the insulin-resistant group (44.9% versus 25.5%; OR 2.38, 95% CI 1.09-5.22; p=0.028), as were acrochordons (31.9% versus 13.7%; OR 2.94, 95% CI

1.15-7.50; p=0.022), intertrigo (27.5% versus 11.8%; OR 2.85, 95% CI 1.04-7.83; p=0.036) and recurrent fungal infection (24.6% versus 9.8%; OR 3.01, 95% CI 1.03-8.77; p=0.039). Keratosis pilaris, acne and hirsutism were more frequent among insulin-resistant children, although these differences did not reach statistical significance. Recurrent bacterial infection was also not significantly associated with insulin resistance (p=0.226). The presence of two or more additional cutaneous markers was significantly more common among children with insulin resistance (53.6% versus 27.5%; OR 3.06, 95% CI 1.42-6.61; p=0.004). Furthermore, insulin-resistant children had a significantly higher mean number of additional cutaneous markers than those without insulin resistance (2.18±1.31 versus 1.29±1.04; MD 0.89, 95% CI 0.47-1.31; t=4.00, p<0.001).

Table 4: Association of acanthosis nigricans and other cutaneous markers with anthropometric and biochemical indicators (N=120)

Indicator	Acanthosis nigricans present (n=73), mean (SD)	Acanthosis nigricans absent (n=47), mean (SD)	Mean difference (95% CI)	Test statistic	*p* value
BMI, kg/m²	28.7 (3.6)	25.9 (3.2)	2.80 (1.56-4.04)	t=4.47	<0.001
BMI Z-score	2.61 (0.48)	2.18 (0.42)	0.43 (0.27-0.59)	t=5.03	<0.001
Waist circumference, cm	91.8 (10.7)	82.6 (9.4)	9.20 (5.46-12.94)	t=4.87	<0.001
Waist-to-height ratio	0.61 (0.07)	0.55 (0.06)	0.06 (0.04-0.08)	t=4.80	<0.001
Systolic blood pressure, mmHg	118.6 (11.8)	112.3 (10.7)	6.30 (2.14-10.46)	t=3.01	0.003
Fasting plasma glucose, mg/dL	96.7 (11.3)	88.9 (8.7)	7.80 (4.18-11.42)	t=4.19	<0.001
Fasting insulin, µIU/mL	24.8 (10.1)	14.6 (6.8)	10.20 (7.15-13.25)	t=6.11	<0.001
HOMA-IR	5.52 (2.43)	3.09 (1.52)	2.43 (1.71-3.15)	t=6.80	<0.001
HbA1c, %	5.68 (0.47)	5.34 (0.39)	0.34 (0.19-0.49)	t=4.30	<0.001
Triglycerides, mg/dL	151.7 (42.6)	127.8 (37.3)	23.90 (9.10-38.70)	t=3.23	0.002
HDL cholesterol, mg/dL	39.6 (7.8)	44.7 (8.1)	-5.10 (-8.05 to -2.15)	t=-3.45	0.001
LDL cholesterol, mg/dL	113.4 (27.9)	102.1 (25.6)	11.30 (1.43-21.17)	t=2.27	0.025

Multivariable logistic-regression analysis of factors independently associated with insulin resistance

Predictor	Adjusted odds ratio (95% CI)	Wald statistic	*p* value
Acanthosis nigricans present	3.28 (1.39-7.74)	7.38	0.007
Moderate-to-severe acanthosis nigricans	3.91 (1.48-10.34)	7.54	0.006
Acrochordons present	2.67 (1.01-7.08)	3.90	0.048
Striae distensae present	2.14 (0.93-4.93)	3.20	0.074
Two or more additional cutaneous markers	2.58 (1.11-6.01)	4.86	0.028
BMI Z-score, per 1-unit increase	2.96 (1.47-5.97)	9.28	0.002
Waist-to-height ratio, per 0.10-unit increase	2.41 (1.27-4.58)	7.21	0.007
Family history of type 2 diabetes	2.36 (1.02-5.46)	4.02	0.045
Age, per 1-year increase	1.08 (0.94-1.24)	1.12	0.290
Male sex	0.91 (0.40-2.08)	0.05	0.824

The regression model was adjusted for age, sex, pubertal stage, BMI Z-score, waist-to-height ratio, family history of diabetes and selected cutaneous markers. Model fit: Hosmer-Lemeshow χ^2=5.47, p=0.706; Nagelkerke R^2 = 0.46; classification accuracy=77.5%.

Children with acanthosis nigricans demonstrated significantly less favourable anthropometric and biochemical profiles than those without it. They had higher mean BMI (28.7±3.6 versus 25.9±3.2 kg/m²), BMI Z-score (2.61±0.48 versus 2.18±0.42), waist circumference (91.8±10.7 versus 82.6±9.4 cm) and waist-to-height ratio (0.61±0.07 versus 0.55±0.06), with all differences being statistically significant at p<0.001. Mean systolic blood pressure was 6.30 mmHg higher among affected children (95% CI 2.14-10.46; p=0.003). Children with acanthosis nigricans also had significantly higher fasting plasma glucose, fasting insulin, HOMA-IR and HbA1c values (all p<0.001). Regarding lipid parameters, they had higher triglyceride concentrations (151.7±42.6 versus 127.8±37.3 mg/dL; p=0.002), lower HDL cholesterol (39.6±7.8 versus 44.7±8.1 mg/dL; p=0.001) and higher LDL cholesterol (113.4±27.9 versus 102.1±25.6 mg/dL; p=0.025).

In the adjusted logistic-regression analysis, the presence of acanthosis nigricans independently increased the odds of insulin resistance by 3.28 times (95% CI 1.39-7.74; p=0.007). Moderate-to-severe acanthosis nigricans showed an even stronger association (aOR 3.91, 95% CI 1.48-10.34; p=0.006). Acrochordons (aOR 2.67; p=0.048), two or more additional cutaneous markers (aOR 2.58; p=0.028), increasing BMI Z-score (aOR 2.96 per unit; p=0.002), increasing waist-to-height ratio (aOR 2.41 per 0.10-unit increase; p=0.007) and a family history of type 2 diabetes (aOR 2.36; p=0.045) were also independent predictors. Striae distensae, age and sex were not independently associated with insulin resistance. The model demonstrated satisfactory calibration (Hosmer-Lemeshow χ^2=5.47, p=0.706), explained 46% of the outcome variation and correctly classified 77.5% of participants.

DISCUSSION

The present cross-sectional study evaluated acanthosis nigricans and other cutaneous markers of insulin resistance among 120 children with obesity. Acanthosis nigricans was detected in 60.8% of participants, and affected children demonstrated greater adiposity, higher fasting insulin and HOMA-IR values, and a higher prevalence of dyslipidaemia. The severity of acanthosis nigricans showed a progressive relationship with BMI Z-score, waist circumference, fasting insulin, HOMA-IR and the prevalence of insulin resistance. Other dermatological findings, particularly acrochordons, striae distensae, intertrigo and recurrent fungal infections, were also more common among children with insulin resistance.

Children with acanthosis nigricans were significantly older than those without the condition. This may reflect the combined effects of increasing adiposity, puberty and physiological reduction in insulin sensitivity during adolescence. Maguolo and Maffeis (2020)[1] reported that acanthosis nigricans becomes more frequent with increasing age and severity of obesity. Pollock et al. (2022)[2] similarly described adolescence, obesity, family history of diabetes and certain ethnic backgrounds as important risk factors. The absence of a significant sex difference in the present study suggests that obesity-related metabolic dysfunction, rather than sex alone, was the principal determinant.

A family history of obesity and type 2 diabetes was significantly more frequent among children with acanthosis nigricans. Calcaterra et al. (2021)[3] recognised familial metabolic risk and excess body weight as important contributors to hyperinsulinaemia and acanthosis nigricans. Genetic susceptibility, shared dietary practices, physical inactivity and a common obesogenic family environment may explain these relationships. The significant association with sedentary behaviour in the present study further supports the role of modifiable lifestyle factors. The Endocrine Society guideline by Styne et al. (2017)[4] and the American Academy of Pediatrics guideline by Hampl et al. (2023)[5] emphasised evaluation of diet, physical activity, screen exposure, family history and metabolic comorbidities in children with obesity.

The prevalence of acanthosis nigricans was 60.8%, with mild, moderate and severe involvement recorded in 24.2%, 22.5% and 14.2% of all participants, respectively. Reported prevalence varies widely because of differences in ethnicity, age, degree of obesity, clinical definitions and study setting. Ng (2017)[6] reported that acanthosis nigricans is highly prevalent among adolescents with obesity and is clinically relevant because of its association with hyperinsulinaemia and type 2 diabetes risk. Videira-Silva et al. (2019)[7] also found acanthosis nigricans to be a useful clinical marker of insulin resistance among adolescents with overweight. The relatively high prevalence in the present study may be attributable to inclusion of children already having obesity and recruitment from a tertiary-care hospital.

The neck was the most common site of acanthosis nigricans, followed by the axillae, knuckles and groin. Hasse et al. (2023)[8] found a high burden of dermatological abnormalities among children and adolescents with obesity and highlighted acanthosis nigricans as one of the most important metabolic skin findings. The posterior neck is easily examined and is generally the most reproducible location for clinical grading. Pollock et al. (2022)[2] recommended careful inspection of the neck, axillae and other intertriginous sites during routine paediatric assessment because early lesions may otherwise be mistaken for poor hygiene or simple hyperpigmentation.

Children with acanthosis nigricans had significantly higher BMI, BMI Z-score, waist circumference and waist-to-height ratio. Koh et al. (2016)[9] similarly observed greater BMI and waist circumference among children with acanthosis nigricans and reported that increasing clinical severity was associated with progressively worsening insulin resistance. Burguete-García et al. (2022)[10] found that a severe quantitative neck score was associated with abdominal obesity, elevated HOMA-IR and hyperlipidaemia among children with obesity. These observations suggest that central adiposity may be particularly important because visceral fat promotes increased free-fatty-acid flux, chronic inflammation and impaired insulin signalling.

Fasting insulin and HOMA-IR were substantially higher among children with acanthosis nigricans.

Insulin resistance was present in 71.2% of affected children compared with 36.2% of those without it, corresponding to more than fourfold higher unadjusted odds. Koh et al. (2016)[9] reported mean fasting insulin values of 24.1 versus 9.8 mU/L and HOMA-IR values of 5.74 versus 2.14 among obese children with and without acanthosis nigricans, respectively. Videira-Silva et al. (2019)[7] likewise found higher fasting insulin and HOMA-IR among adolescents with acanthosis nigricans. These findings support the biological explanation that compensatory hyperinsulinaemia activates insulin-like growth factor receptors in keratinocytes and fibroblasts, resulting in epidermal proliferation, hyperkeratosis and papillomatosis.

A particularly important finding was the graded relationship between acanthosis nigricans severity and metabolic dysfunction. Mean HOMA-IR increased from 3.09 among children without acanthosis nigricans to 7.44 among those with severe disease. The prevalence of insulin resistance increased simultaneously from 36.2% to 82.4%, with an OR of 2.34 for each successive severity category. Koh et al. (2016)[9] reported an almost identical progression in HOMA-IR from 2.15 in children without acanthosis nigricans to 4.15 in mild disease and 7.22 in severe disease. The findings of Burguete-García et al. (2022)[10] also demonstrated that more severe neck involvement was associated with abdominal obesity, insulin resistance and hyperlipidaemia. Thus, grading acanthosis nigricans may offer greater metabolic information than recording its presence alone.

Dyslipidaemia was significantly more frequent among affected children. They had higher triglyceride and LDL-cholesterol levels and lower HDL-cholesterol levels. Pires et al. (2015)[11] demonstrated that insulin resistance among children with obesity was accompanied by dyslipidaemia and adverse cardiovascular alterations. Chedjou-Nono et al. (2017)[12] also documented clustering of abdominal obesity, insulin resistance, hypertension and lipid abnormalities in paediatric obesity. Karadağ et al. (2018)[13] described acanthosis nigricans as a visible component of the broader metabolic-syndrome phenotype. These findings indicate that a child presenting with acanthosis nigricans should not be assessed only for abnormal glucose metabolism but should also undergo blood-pressure and lipid evaluation.

The current study also identified higher systolic blood pressure, fasting plasma glucose, HbA1c and adverse lipid measurements among children with acanthosis nigricans. Martinez-Rojano et al. (2016)[14] similarly reported that acanthosis nigricans was associated with cardiovascular risk factors among Mexican children with obesity. The coexistence of central obesity, elevated blood pressure, hyperglycaemia and dyslipidaemia indicates early cardiometabolic clustering and strengthens the value of acanthosis nigricans as a readily visible sign warranting comprehensive metabolic evaluation.

Cutaneous markers other than acanthosis nigricans were common, occurring in 76.8% of insulin-resistant children compared with 52.9% of children without insulin resistance. Hasse et al. (2023)[8] reported a high prevalence of multiple skin disorders among paediatric patients with obesity, with the number of dermatological abnormalities increasing with obesity severity. Obesity can affect the skin through hyperinsulinaemia, altered androgen activity, inflammation, excessive sweating, mechanical friction, impaired skin-barrier function and changes in immune defence.

Striae distensae were significantly associated with insulin resistance in bivariable analysis. Their development probably reflects rapid stretching of the skin, reduced dermal connective-tissue integrity and hormonal influences. However, striae did not remain independently significant after adjustment, indicating that their association may be mediated mainly through the severity of adiposity. Thus, striae should be interpreted as a marker of rapid weight gain and obesity severity rather than as a specific indicator of insulin resistance.

Acrochordons were approximately three times more frequent among insulin-resistant children and remained independently associated with insulin resistance. Das et al. (2020)[15] reviewed the relationship between acanthosis nigricans, hyperinsulinaemia and associated cutaneous findings, including skin tags. The presence of acrochordons in a child is relatively unusual and, particularly when multiple lesions coexist with acanthosis nigricans, may justify metabolic screening.

Intertrigo and recurrent fungal infections were also significantly more common among insulin-resistant participants. Increased skin-fold depth, moisture retention, friction, impaired barrier function and hyperglycaemia may promote Candida and dermatophyte growth. A lower frequency of recurrent bacterial infection and its statistically non-significant association may reflect limited statistical power or differences in hygiene, exposure and previous treatment. Keratosis pilaris, acne and hirsutism showed higher frequencies among insulin-resistant children but did not achieve statistical significance. Acne and hirsutism may be influenced by puberty, androgen concentrations and polycystic ovarian syndrome, while keratosis pilaris is common even in

otherwise healthy children; these factors may have reduced their specificity for insulin resistance.

Children with insulin resistance had a significantly greater total number of additional cutaneous markers, and the presence of two or more markers remained independently associated with insulin resistance. This suggests a cumulative clinical effect: although an individual lesion may have limited specificity, the coexistence of several obesity-related skin findings may indicate a higher metabolic-risk phenotype. A complete skin examination may consequently provide more useful information than screening for acanthosis nigricans alone.

After adjustment for potential confounders, acanthosis nigricans remained independently associated with insulin resistance, with an adjusted OR of 3.28. Moderate-to-severe disease showed a stronger association, with an adjusted OR of 3.91. Lopez-Alvarenga et al. (2020)[16] described acanthosis nigricans as a composite clinical marker of obesity and insulin resistance, while Sayarifard et al. (2021)[17] reported a relationship between acanthosis nigricans and metabolic syndrome among children with obesity. These findings support the use of acanthosis nigricans as an accessible risk-stratification sign, although it should not be treated as a diagnostic test in isolation.

BMI Z-score and waist-to-height ratio were independent predictors of insulin resistance. The stronger contribution of central adiposity supports the use of waist-based measurements alongside BMI. Weihe and Weihrauch-Blüher (2019)[18] noted that cardiometabolic complications in paediatric obesity depend not only on overall weight but also on fat distribution and associated metabolic dysfunction. Reinehr (2018)[19] similarly highlighted that insulin resistance, dyslipidaemia and hypertension may already be established during childhood.

A family history of type 2 diabetes independently increased the likelihood of insulin resistance, whereas age and sex were not independently significant. This suggests that the unadjusted association with older age was partly explained by pubertal development and increasing adiposity. The International Society for Pediatric and Adolescent Diabetes recommendations by Zeitler et al. (2018)[20] identified obesity, family history of diabetes and signs of insulin resistance, including acanthosis nigricans, as important considerations for type 2 diabetes screening in young people.

The regression model demonstrated acceptable calibration and correctly classified 77.5% of participants. Nevertheless, clinical findings should complement rather than replace biochemical assessment. The American Diabetes Association (2024)[21] recommends risk-based screening for prediabetes and type 2 diabetes among children with overweight or obesity who have additional risk factors. Similarly, the Indian Academy of Pediatrics guideline reviewed by Gaikwad and Kumar (2023)[22] emphasised early, structured assessment and management of childhood obesity and its complications.

Overall, the findings demonstrate that acanthosis nigricans especially moderate-to-severe disease and the coexistence of multiple cutaneous abnormalities can serve as visible indicators of an adverse metabolic profile. Routine examination of the neck and flexural areas is simple, inexpensive and feasible in paediatric and dermatology outpatient settings. Detection should initiate anthropometric assessment, blood-pressure measurement and appropriate biochemical screening, followed by family-centred lifestyle intervention.

CONCLUSION

Acanthosis nigricans was present in approximately three-fifths of children with obesity and was strongly associated with central adiposity, hyperinsulinaemia, insulin resistance, dyslipidaemia and other adverse cardiometabolic indicators. Increasing severity of acanthosis nigricans was accompanied by progressive increases in BMI Z-score, waist circumference, fasting insulin, HOMA-IR and the prevalence of insulin resistance.

Acrochordons, striae distensae, intertrigo and recurrent fungal infections were more common among insulin-resistant children, while the presence of multiple cutaneous markers further increased the likelihood of insulin resistance. Acanthosis nigricans, particularly moderate-to-severe disease, remained an independent predictor after adjustment for adiposity and familial risk factors. Therefore, a careful skin examination can provide a simple and inexpensive opportunity to identify children with obesity who require comprehensive metabolic screening and early lifestyle intervention.

LIMITATIONS OF STUDY

1. The cross-sectional design established associations but could not determine temporal or causal relationships between obesity, insulin resistance and cutaneous manifestations.
2. The study was conducted at a single tertiary-care hospital; therefore, referral bias may have resulted in the inclusion of children with more severe obesity or metabolic abnormalities.
3. The relatively small sample size limited the precision of subgroup analyses, particularly those

involving hirsutism, recurrent infections and individual severity categories.

4. Participants without obesity and normal-weight healthy controls were not included, limiting comparison with the general paediatric population.
5. Insulin resistance was estimated using HOMA-IR rather than the hyperinsulinaemic-euglycaemic clamp, which is considered the reference method.
6. HOMA-IR cut-offs vary according to age, sex, ethnicity and pubertal status; therefore, some misclassification may have occurred.
7. Acanthosis nigricans severity and other cutaneous findings were assessed clinically and were subject to observer variation, although standard criteria were used.
8. Dietary intake, physical activity and screen exposure were based partly on participant or parental recall and were therefore susceptible to recall and social-desirability bias.
9. Potential confounders such as detailed pubertal hormone levels, body-fat percentage, socioeconomic status and genetic predisposition could not be assessed comprehensively.
10. The study did not include follow-up to determine whether weight reduction or improvement in insulin sensitivity resulted in regression of the cutaneous lesions.

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