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CUTANEOUS MARKERS OF INSULIN RESISTANCE AND METABOLIC SYNDROME: DIAGNOSTIC VALUE AND IMPLICATIONS FOR EARLY INTERVENTION - A NARRATIVE REVIEW

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ABSTRACT

Background: Insulin resistance and metabolic syndrome may remain clinically silent, while selected cutaneous findings are visible during routine examination. Their associations with metabolic abnormalities vary, and they are not diagnostic tests.
Objective: To summarize evidence linking selected skin findings with insulin resistance and metabolic syndrome and outline a cautious approach to metabolic assessment in dermatology and primary care.
Methods: A targeted narrative search of PubMed, Google Scholar and reference lists identified English-language guidelines, reviews, observational studies and relevant case series available through 31 October 2026. Search concepts covered insulin resistance, metabolic syndrome, acanthosis nigricans, acrochordons, xanthomas and related dermatoses. Evidence was synthesized qualitatively.
Results: Acanthosis nigricans has the clearest association with hyperinsulinemia and obesity-related metabolic risk, but diagnostic performance varies across populations and study designs. Multiple acrochordons are associated with dysglycemia and metabolic-syndrome components but are nonspecific. Eruptive xanthomas may signal severe hypertriglyceridemia. Hidradenitis suppurativa and psoriasis are associated with cardiometabolic comorbidity, while acne, hirsutism and female-pattern hair loss require consideration of hyperandrogenism and polycystic ovary syndrome. No single skin sign establishes or excludes insulin resistance or metabolic syndrome.
Conclusions: Selected skin findings can prompt risk assessment and guideline-based testing, particularly alongside central adiposity or other risk factors. They are clinical cues, not validated stand-alone tests. Prospective studies with standardized definitions are needed.

KEYWORDS
Acanthosis Nigricans, Insulin Resistance, Metabolic Syndrome, Obesity, Skin Manifestations

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1. Introduction

Insulin resistance (IR) describes reduced biological responsiveness to insulin in one or more insulin-sensitive tissues. It often precedes dysglycemia and may coexist with central adiposity, dyslipidemia, elevated blood pressure and metabolic dysfunction-associated steatotic liver disease (MASLD). Metabolic syndrome is a cluster of interrelated cardiometabolic risk factors rather than a single disease, and its prevalence and definition depend on the criteria used [1-4]. These abnormalities may remain asymptomatic until type 2 diabetes or vascular disease develops.

The skin is readily examined during routine care and may reflect endocrine, metabolic, inflammatory or mechanical processes. Acanthosis nigricans (AN), acrochordons, eruptive xanthomas and several inflammatory or androgen-related dermatoses have been associated with metabolic abnormalities [5-9]. The strength and meaning of these associations differ substantially. A lesion may be a correlate of obesity, hyperinsulinemia, androgen excess, chronic inflammation or established diabetes; these mechanisms are not interchangeable.

An association does not establish diagnostic accuracy, causality or clinical utility. Skin signs are influenced by age, ethnicity, skin phototype, medication exposure, genetic susceptibility and local friction. Conversely, the absence of a cutaneous sign cannot reassure against dysglycemia. This review summarizes the principal findings, evaluates the limits of the evidence and proposes a practical approach to metabolic assessment when potentially relevant skin signs are observed.

1.1. Metabolic concepts relevant to dermatologic practice

Insulin resistance is a pathophysiological state rather than a diagnosis that can be inferred from one universally accepted routine test. Reduced insulin action in skeletal muscle, liver and adipose tissue may initially be compensated for by increased pancreatic insulin secretion. Over time, this compensation may become inadequate, leading to impaired glucose regulation and, in some individuals, type 2 diabetes. The timing and extent of this progression differ according to genetics, age, adiposity, physical activity, medication exposure and other determinants [10,11,40]. This distinction matters in dermatology because a visible lesion may coexist with hyperinsulinemia long before glucose-based diagnostic criteria are met, while another patient with diabetes may have no recognizable skin marker.

Metabolic syndrome is a clinical construct used to identify a clustering of cardiometabolic risk factors. Common definitions include central adiposity, elevated triglycerides, reduced HDL cholesterol, raised blood pressure and elevated fasting glucose, but they differ in the role of waist circumference and the thresholds applied to particular populations [1-4,38,39]. The syndrome helps organize risk assessment; it does not replace the evaluation of each component or establish a single causal pathway. Consequently, the phrase “skin marker of metabolic syndrome” should be understood as shorthand for an association with one or more components, not as a dermatologic diagnostic criterion.

The distinction between association, prediction and diagnosis is central to interpreting the literature. An association indicates that a finding and an outcome occur together more often than expected in a studied population. Prediction requires evidence that the finding improves estimation of future or current risk beyond information already available, such as age, waist circumference and family history. Diagnosis requires a defined clinical or laboratory standard. Many dermatology studies establish association but do not test whether examining the skin improves screening performance or patient outcomes [14-25].

1.2. Why visible findings may matter and where the inference stops

Dermatology has a practical opportunity to identify patients who may not otherwise seek preventive care. A clinician may observe acanthosis nigricans during an examination for an unrelated complaint, or a patient may seek treatment for skin tags, hidradenitis suppurativa or psoriasis. Such encounters can create a reasonable moment to ask about established risk factors and prior screening. This opportunity is particularly relevant when primary-care access is limited, although it should complement rather than displace routine preventive services.

The biological links are heterogeneous. Acanthosis nigricans may reflect hyperinsulinemia and signaling through insulin-like growth factor pathways; skin tags may be related to a mixture of age, friction, adiposity and metabolic factors; eruptive xanthomas reflect lipid deposition in the setting of marked triglyceride elevation; and psoriasis or hidradenitis suppurativa are chronic inflammatory diseases associated with cardiometabolic comorbidity [5,12,13,21-34]. These conditions should not be grouped as if they shared one mechanism or the same predictive value.

Clinical interpretation also needs to account for the patient’s context. Skin phototype can affect the visibility of erythema and pigmentary change; age affects the baseline prevalence of acrochordons; pregnancy, puberty and menopause alter metabolic and hormonal physiology; and medications may contribute to insulin resistance or mimic particular lesions. A single photo or visual description may not capture texture, distribution, tempo of onset or associated symptoms. These factors can lead to both over-recognition and under-recognition of a proposed marker.

The aim of this narrative review was to evaluate the diagnostic value of selected cutaneous findings associated with IR and metabolic syndrome, distinguish stronger from weaker associations, and describe when these findings may reasonably prompt cardiometabolic risk assessment in dermatology and primary care.

2. Methodology

A targeted narrative search was conducted in PubMed, Google Scholar, with additional citation searching of relevant reviews and guidelines. Searches covered records available through 31 October 2026. Combinations of the following terms were used: “insulin resistance,” “metabolic syndrome,” “acanthosis nigricans,” “acrochordons,” “skin tags,” “eruptive xanthomas,” “hidradenitis suppurativa,” “psoriasis,” “acne,” “hirsutism,” “androgenetic alopecia,” “obesity,” “prediabetes,” and “type 2 diabetes.”

English-language clinical practice guidelines, consensus statements, systematic reviews, observational studies and clinically relevant case series were considered. Priority was given to studies that described their populations, metabolic outcomes and lesion definitions. Case reports were used only to illustrate uncommon or urgent presentations. Findings were synthesized qualitatively; no pooling was attempted because populations, definitions of insulin resistance, metabolic-syndrome criteria, lesion ascertainment and study designs differed. This narrative review did not use a registered systematic-review protocol, formal risk-of-bias tool or meta-analysis.

2.1. Scope and analytic framework

The review focused on cutaneous findings encountered in general dermatology and primary care that have been linked in clinical literature to insulin resistance, dysglycemia, dyslipidemia or metabolic syndrome. The findings were grouped by the type of clinical signal they may represent: (1) findings most often associated with hyperinsulinemia or adiposity-related risk, (2) lesions that may indicate a specific metabolic abnormality requiring timely assessment, (3) inflammatory disorders associated with cardiometabolic comorbidity, and (4) androgen-related findings that may prompt evaluation for polycystic ovary syndrome or another endocrine disorder. This framework was used to avoid treating all associations as diagnostically equivalent.

Evidence was interpreted in relation to study design and population. Cross-sectional studies can identify co-occurrence but cannot establish temporal order. Case-control studies can compare selected groups but may be vulnerable to selection bias. Studies based in obesity clinics, dermatology referral centers or specialist endocrine services may report stronger associations than would be seen in unselected community practice. Reviews and guidelines were used to contextualize primary studies, while their conclusions were not treated as independent primary data.

2.2. Interpretation of outcomes and limitations of the search

Metabolic outcomes were not considered interchangeable. A study reporting fasting insulin or HOMA-IR addresses a surrogate estimate of insulin resistance; a study reporting impaired glucose tolerance or diabetes addresses glycemic status; and a study using metabolic-syndrome criteria evaluates a composite defined by a particular consensus. An association with one outcome does not automatically establish an association with all the others. Where studies used different thresholds or methods, findings were described qualitatively rather than combined numerically [1-4,10,11].

The search was targeted toward influential and clinically relevant evidence and was not designed to guarantee exhaustive retrieval. Database coverage, indexing, publication language and citation practices may have affected which records were identified. The manuscript does not present a screening flow diagram or counts of records at each selection stage; the findings should therefore be interpreted as a narrative synthesis rather than a systematic review.

3. Results

3.1. Insulin Resistance and Interpretation of Cutaneous Associations

Clinical meaning of surrogate measures

For many patients, the actionable question is whether they meet accepted criteria for prediabetes or diabetes, have dyslipidemia or hypertension, or have an overall risk profile that warrants intervention. Glucose-based tests, blood pressure measurement and lipid assessment are linked to established prevention and treatment pathways [38-40]. A skin sign may contribute to clinical suspicion, but testing decisions should remain aligned with relevant age-based and risk-based recommendations.

3.2. Acanthosis Nigricans

AN typically presents as symmetric, velvety or papillomatous hyperpigmented thickening, most often on the posterior neck, axillae and other flexures. It may also affect knuckles or less typical sites. In obesity-associated AN, hyperinsulinemia is thought to activate insulin-like growth factor pathways in keratinocytes and dermal fibroblasts, contributing to epidermal proliferation [5,12,13]. The clinical appearance can be subtle, particularly in darker skin phototypes, and post-inflammatory pigmentation or other flexural dermatoses may mimic it.

Among the cutaneous findings reviewed here, AN has the most established relationship with hyperinsulinemia and obesity-related metabolic risk. Observational studies in children and adults have reported associations with fasting insulin, HOMA-derived indices, glucose intolerance, triglycerides and other components of metabolic syndrome [14-19]. However, results are not uniform. Several studies are cross-sectional, recruit from obesity or specialty clinics, or use different definitions of IR and AN severity. Diagnostic values reported in one population cannot be assumed to apply to another.

A 2026 systematic review identified a large body of literature relating AN to metabolic-syndrome components, but included a substantial number of case reports and heterogeneous populations; its pooled descriptive proportions should not be interpreted as the predictive value of AN in an unselected primary-care population [20]. AN is therefore a clinically useful prompt for risk assessment, not a stand-alone test. Abrupt-onset, extensive or atypical AN, particularly with mucosal involvement, tripe palms, unexplained weight loss or rapid progression in an adult, requires consideration of alternative causes, including medication effects, endocrinopathies and rarely paraneoplastic disease [5,12].

Morphology, distribution and differential diagnosis

Differential diagnosis includes post-inflammatory hyperpigmentation, frictional change, confluent and reticulated papillomatosis, terra firma-forme dermatosis and other flexural disorders. Cleansing or examination of surface change may assist in selected cases, but clinical context remains essential. In darker phototypes, erythema may be less conspicuous and hyperpigmentation may be the most apparent feature; palpation for texture and comparison with nearby skin can therefore be useful. Misclassification can distort both clinical risk assessment and research estimates.

In children and adolescents, AN should be interpreted in relation to growth, puberty, family history, adiposity and ethnicity. Pediatric studies have reported associations with hyperinsulinemia and diabetes risk factors, but their referral settings and age distributions differ [15–19]. Puberty itself changes insulin sensitivity, and adult thresholds cannot simply be transferred to children. When the finding is present, the clinician should apply pediatric screening guidance and avoid stigmatizing language; the lesion is a clinical observation, not evidence of non-adherence or personal fault.

3.3. Acrochordons

Acrochordons (skin tags) are common benign soft papules, often found on the neck, axillae and groin. Their prevalence increases with age and may be higher with obesity and friction. Case-control studies have reported associations between multiple acrochordons and impaired glucose tolerance, diabetes, dyslipidemia, hypertension or metabolic syndrome [21-25]. Other studies have found weaker or inconsistent associations after adjustment for age and adiposity.

Count, size and distribution have been proposed as risk indicators, but there is no validated lesion threshold for ordering laboratory testing. A few incidental skin tags in an older adult have low specificity. A high burden of lesions in a younger person, particularly alongside AN or central adiposity, may reasonably lead to a review of standard cardiometabolic risk factors. Removal of acrochordons is a separate cosmetic or symptomatic decision and does not address metabolic risk.

Burden of lesions and confounding

The proposed relationship between acrochordons and metabolic abnormalities is biologically and epidemiologically plausible but not sufficiently specific to support diagnosis. Skin tags become more common with increasing age and can be influenced by friction and anatomic location. Obesity may contribute to both mechanical friction and metabolic risk, creating confounding when studies compare people with and without lesions. A higher lesion count may be associated with dysglycemia in some clinical samples, but no universally accepted number, size or distribution has been validated as a threshold for laboratory testing [21-25].

When a patient presents for removal of numerous or newly noticed lesions, a brief review of established risk factors may be reasonable, particularly if the patient is younger than expected for the lesion burden or has

central adiposity, a family history of diabetes or prior gestational diabetes. The decision to test should be based on the complete risk profile and applicable guidelines. This distinction helps prevent common benign lesions from becoming a source of alarm while still allowing them to prompt useful preventive care.

3.4. Eruptive Xanthomas and Other Lipid-Associated Signs

Eruptive xanthomas are crops of small yellow-red papules, often with an erythematous halo, commonly on extensor surfaces and buttocks. They may occur with marked hypertriglyceridemia, sometimes in the setting of uncontrolled diabetes, genetic lipid disorders or secondary causes such as alcohol use and medications [26,27]. Their clinical importance differs from AN or acrochordons: they can signal severe dyslipidemia that warrants prompt laboratory assessment and clinical management because of associated pancreatitis risk.

Distinguishing a high-priority lipid signal

A differential diagnosis is still needed. Papular lesions may represent other inflammatory or infiltrative disorders, and the appearance of xanthomas can vary with skin tone and stage. Clinical assessment should include medication and alcohol history, diabetes status, family history and symptoms that could indicate pancreatitis. The skin finding is a clue to a biochemical abnormality; laboratory confirmation is needed to characterize the lipid profile and guide care.

Xanthelasma consists of yellowish plaques, usually on the eyelids, while arcus corneae is a peripheral corneal opacity. Both can coexist with lipid abnormalities, but their significance depends on age and cardiovascular context. They should not be treated as equivalent to eruptive xanthomas or as specific evidence of insulin resistance [28].

3.5. Inflammatory Dermatoses Associated with Cardiometabolic Risk

For patients with HS or psoriasis, clinicians can use routine opportunities to assess weight trajectory, blood pressure, smoking, family history and guideline-based indications for glucose and lipid testing. Screening should be framed without stigma and should not imply that body weight is the sole cause of inflammatory disease.

Inflammation, shared risk and clinical pathways

Hidradenitis suppurativa is a relapsing inflammatory disorder with substantial effects on pain, function and quality of life. Observational studies and reviews describe higher prevalence of obesity, smoking, diabetes, dyslipidemia and metabolic syndrome in affected populations [29–31]. These patterns support attention to comorbidity, but they do not show that HS is a direct marker of insulin resistance in an individual patient. Disease severity, duration, treatment exposure and associated behaviors may influence observed relationships, and inflammation may interact with metabolic factors in more than one direction.

Psoriasis likewise has established associations with obesity and cardiometabolic disease, and chronic systemic inflammation may contribute to shared risk [32-34]. The presence of psoriasis can therefore be an opportunity to review cardiovascular risk factors and coordinate preventive care. However, psoriasis is common and heterogeneous; its presence cannot be used to diagnose diabetes or metabolic syndrome, and its severity does not substitute for measuring blood pressure, glucose or lipids.

A practical approach is to integrate risk review into existing disease care rather than create a separate dermatology-specific metabolic diagnosis. Clinicians can document blood pressure when available, ask whether the patient has a primary-care clinician, review smoking and family history, and encourage appropriate screening. Referral pathways should be clear when abnormalities are identified. The goal is to connect patients with established prevention and treatment, not to suggest that treatment of the skin disease alone will resolve cardiometabolic risk.

3.6. Androgen-Related Findings: Acne, Hirsutism and Female-Pattern Hair Loss

Acne, hirsutism and female-pattern hair loss may be associated with polycystic ovary syndrome (PCOS) and hyperandrogenism, in which IR is common but not universal [35-37]. These signs have distinct dermatologic and endocrine differentials. Acne alone is common and has poor specificity for IR. Hirsutism should be assessed using an appropriate clinical method and in relation to menstrual history, medication use and signs of virilization. Sudden onset, rapid progression or virilization warrants timely evaluation for causes beyond PCOS.

In a patient with suspected PCOS, metabolic assessment should follow current diagnostic and screening guidance for PCOS and diabetes risk rather than rely on skin signs or fasting insulin alone [36,37]. This avoids conflating androgen excess with IR and reduces unnecessary testing.

Avoiding over-interpretation of common skin findings

The 2023 international PCOS guideline emphasizes a comprehensive diagnostic approach and appropriate assessment of metabolic risk in patients with PCOS [36]. Dermatologic symptoms may contribute to recognizing a possible syndrome, but clinicians should not diagnose PCOS from acne or hair loss alone. Conversely, absence of a skin manifestation does not exclude PCOS. Sudden virilization, deepening of the voice or rapid progression of hirsutism warrants timely evaluation for causes beyond typical PCOS.

3.7. A Practical Clinical Approach

A stepwise assessment in routine care

A useful first step is to define the skin finding precisely: morphology, anatomic distribution, number of lesions, onset, progression, symptoms and relevant photographs where clinically appropriate. The clinician should consider whether the finding is characteristic, whether common mimics are plausible, and whether the pattern is new or longstanding. This avoids anchoring on a metabolic explanation before establishing the dermatologic diagnosis.

The second step is to distinguish the type of signal. Acanthosis nigricans or a high burden of acrochordons may prompt review of general cardiometabolic risk; eruptive xanthomas may call for prompt assessment of triglycerides; HS and psoriasis support attention to comorbidity screening; and androgen-related findings may warrant an endocrine history and evaluation. This categorization helps match the urgency and type of assessment to the likely clinical issue rather than ordering the same panel for every patient.

The third step is to assess established risk factors. Relevant information can include age, body mass index and waist circumference when appropriate, blood pressure, family history of diabetes or premature cardiovascular disease, previous gestational diabetes, physical activity, smoking, medication use and known dyslipidemia or dysglycemia. A patient's social circumstances and access to primary care may affect whether recommended testing has been completed. The skin finding should be integrated with this information rather than considered in isolation.

The encounter should also account for whether the patient is already receiving care for metabolic disease. If diabetes, dyslipidemia or hypertension is known, the skin finding may be relevant to treatment context or complications, but it does not independently indicate that control is inadequate. If prior results are available, reviewing them may be more useful than repeating tests. In patients without a primary-care clinician, dermatology teams can explain the reason for referral and help direct the patient to an appropriate service, while avoiding assumptions about access, adherence or health literacy.

Clinicians should be alert to the possibility that the same cutaneous presentation has more than one explanation. For example, flexural hyperpigmentation may coexist with obesity-related insulin resistance but can also reflect chronic irritation or another dermatosis; hair loss may be influenced by iron deficiency, thyroid disease, medication or genetic patterning in addition to androgen excess. A metabolic association should not prematurely close the dermatologic or endocrine differential. When morphology is atypical, symptoms are progressive, or the history suggests an alternative cause, targeted evaluation should follow the clinical presentation rather than a presumed metabolic pathway.

The fourth step is to select tests based on clinical guidance and the suspected abnormality. Depending on age, symptoms and risk, glucose assessment may involve fasting plasma glucose, HbA1c or an oral glucose tolerance test; lipid assessment may be appropriate for suspected xanthomas or as part of preventive care [38-40]. HOMA-IR and fasting insulin are not required to diagnose diabetes or metabolic syndrome and should not be ordered reflexively because a patient has AN or skin tags [10,11]. If results are abnormal, follow-up and treatment should occur through appropriate clinical pathways.

Finally, document the reasoning and follow-up plan. A concise record can state the observed morphology, associated risk factors reviewed, tests offered or arranged, and the plan for communicating results. If testing is not indicated or is declined, the rationale and an opportunity for future reassessment can be documented. This makes the process transparent and reduces the chance that a "metabolic marker" label is interpreted as a confirmed diagnosis.

Person-centered communication and stigma

The language used to discuss metabolic risk can influence whether a patient feels respected and is willing to engage in preventive care. Explain that some skin findings occur more often alongside certain metabolic abnormalities, while emphasizing that they are neither proof of disease nor a measure of a person's behavior

or worth. Avoid implying that body weight alone caused a skin disorder. Ask permission before discussing weight, use neutral terminology and focus on actionable health measures and the patient's goals.

A brief, non-alarmist explanation may be sufficient in many consultations: "This skin change is sometimes seen together with changes in blood sugar or cholesterol. It does not diagnose those problems, but given your other risk factors, it may be sensible to check whether routine screening is up to date." The wording should be adapted to the patient's situation and preferences. Such communication preserves uncertainty, gives a clear rationale for testing and avoids implying that a visible lesion predicts an inevitable outcome.

Patients may reasonably ask whether removing a lesion or treating an inflammatory dermatosis will reduce metabolic risk. The answer depends on the condition: removal of an acrochordon is a local procedure and does not treat dysglycemia; treatment of HS or psoriasis addresses skin disease and may improve quality of life, but it should not replace cardiovascular risk assessment. Clear explanations can prevent cosmetic treatment from being mistaken for metabolic treatment and support coordination with primary care.

4. Discussion

The literature supports a graded interpretation of skin findings. AN has the clearest association with hyperinsulinemia and obesity-related risk. Multiple acrochordons may be a useful additional clue, but estimates are less consistent and are affected by age and adiposity. Eruptive xanthomas have a high clinical signal for severe triglyceride elevation in the appropriate morphology, although they are uncommon. HS and psoriasis identify populations with increased cardiometabolic comorbidity, but they are inflammatory diseases rather than specific markers of IR. Acne, hirsutism and female-pattern hair loss require interpretation through an androgen and PCOS framework.

The same clinical observation can also have different implications depending on baseline risk. Acanthosis nigricans in a patient with central adiposity, a family history of diabetes and prior gestational diabetes provides a different pretest context from subtle flexural pigmentation in a low-risk patient. Similarly, skin tags in an older adult may be an age-associated finding, whereas numerous new lesions in a younger patient may justify a closer review of risk factors. Such examples illustrate why lesion-based decisions should be made within an overall clinical assessment rather than through a universal testing threshold.

The term "diagnostic value" should be used cautiously. Many studies report association or prevalence rather than sensitivity, specificity, likelihood ratios or improvement in patient outcomes after skin-based case finding. Referral and specialty-clinic samples may overestimate the association. Cross-sectional designs cannot establish whether a lesion precedes metabolic disease or reflects a shared cause. Differences in skin phototype and examination methods may also affect lesion recognition, especially for pigmentary change. The 2026 systematic review of AN underscores both the depth of available literature and its heterogeneity [20].

A further interpretive issue is whether a visible marker adds information beyond routinely available clinical data. If age, waist circumference, blood pressure, family history and prior glycemic results already identify a patient as eligible for screening, the skin finding may reinforce the need to complete testing but may not change the decision itself. Its incremental value could be greater in patients whose risk has not previously been recognized or whose access to preventive care is limited. This hypothesis should be tested directly by comparing usual risk assessment with risk assessment that includes a standardized skin examination. Outcomes should include not only detection rates but also patient understanding, completion of follow-up, clinical actionability and any unintended effects.

The clinical value of a cutaneous marker may lie less in diagnosing IR itself than in creating an opportunity to identify established, actionable risks. A focused skin examination can be incorporated into routine care, but no evidence currently supports using a dermatologic sign as a substitute for validated diabetes or cardiovascular risk assessment. Research should prospectively test whether standardized skin assessment improves screening uptake, diagnostic yield or outcomes compared with usual care. Studies should prespecify age and skin-phototype strata, use reproducible lesion definitions, distinguish association from prediction, and report calibration and diagnostic performance in representative populations.

The evidence base has also begun to consider whether cutaneous changes could be followed over time during weight or metabolic treatment [41]. This is an interesting hypothesis, but it should be separated from the question of screening. A lesion that improves after weight reduction or improved glycemic control may be a responsive clinical feature without serving as a validated surrogate for cardiometabolic outcomes. To establish a monitoring role, studies would need to define the lesion outcome reproducibly, assess whether change tracks meaningful metabolic improvement independently of other factors, and determine whether the information changes management. Until then, improvement in skin appearance should not be used to infer that diabetes or cardiovascular risk has resolved.

Strengths and limitations of the available evidence

A strength of the literature is that it spans several levels of evidence: mechanistic work, clinical cohorts, cross-sectional studies, case-control comparisons, systematic reviews and professional guidelines. This supports a clinically nuanced synthesis rather than a single-marker approach. The evidence is most useful when each lesion is considered in relation to a defined outcome and the population studied. It is less useful when a study's association is generalized to all patients or converted into a diagnostic claim without validation.

Important limitations recur across the evidence base. Definitions of insulin resistance and metabolic syndrome vary; lesion definitions and severity scales are not consistently applied; and studies may differ in age, ethnicity, skin phototype and referral setting. Some studies use small samples or rely on self-reported diagnoses. Cross-sectional designs cannot show whether the skin change preceded the metabolic abnormality. Residual confounding by adiposity, age, medication use and socioeconomic factors may remain even after statistical adjustment [14-25, 29-37]. These issues make pooled estimates difficult to translate into an individual patient's probability of disease.

A related narrative review published in 2026 examined dermatologic manifestations in people with morbid obesity, including acanthosis nigricans, acrochordons, inflammatory disorders and possible changes during metabolic treatment [41]. Its scope is therefore close to the present topic, and its conclusions likewise caution that associations do not establish validated diagnostic or prognostic tools. The present review addresses a somewhat different clinical question: how individual findings should be interpreted when considering insulin resistance or metabolic syndrome in dermatology and primary care, including the distinction between general risk cues and lesions that may signal a specific urgent abnormality. It also considers eruptive xanthomas, lipid-associated signs and androgen-related presentations, and emphasizes the limits of routine insulin measures and lesion-based screening. This distinction is one of emphasis rather than an entirely separate evidence base; both reviews draw on overlapping literature and should be read as complementary syntheses.

There is also a gap between detecting association and demonstrating clinical utility. To establish utility, a study would need to show that including a skin examination improves on routine risk assessment for example, by identifying additional patients who meet diagnostic criteria, increasing completion of appropriate testing, or improving outcomes without causing excessive false-positive workups or stigma. The reviewed studies generally do not provide this level of evidence. Accordingly, the review supports a cautious clinical prompt, not a formal screening algorithm based on lesion counts or visual severity.

Potential harms of over-interpreting cutaneous findings deserve attention. False-positive labeling may result in unnecessary laboratory investigations, anxiety, repeated follow-up or weight-related stigma. False reassurance is also possible if a patient without visible markers is assumed to have low metabolic risk. Because many people with dysglycemia have no specific skin sign, a skin-focused approach cannot replace preventive screening based on age and established risk factors [38-40]. The expected benefit of a marker-based prompt is therefore greatest when it helps close a gap in care without creating a new diagnostic label.

Authors should also distinguish between screening prompted by a clinical finding and population screening. The former is an individualized response to the examination and history; the latter requires evidence that a program's benefits outweigh its harms across a defined population. The current literature supports careful case finding, but it does not establish a population-wide skin-screening program.

Priorities for future research

Prospective, multicenter studies should recruit representative primary-care and dermatology populations and prespecify standardized diagnostic criteria for skin findings and metabolic outcomes. Investigators should report age, sex, ethnicity and skin phototype, and should use reproducible examination protocols. For proposed prediction models, studies should report sensitivity, specificity, likelihood ratios, calibration and incremental performance beyond routine risk factors. External validation is needed before a marker-based score is used in practice.

For clinical implementation, studies should also report the feasibility and acceptability of skin-based assessment. Time required for examination, training needs, inter-rater reliability and patient preferences may determine whether an approach can be adopted in busy practices. If a lesion score is proposed, investigators should assess whether different clinicians can apply it consistently and whether performance remains stable across skin phototypes and clinical settings. A statistically significant association alone would not establish that a score should be incorporated into routine practice.

Intervention studies should evaluate the complete care pathway rather than knowledge alone. Relevant outcomes include whether a patient completes recommended testing, whether clinically meaningful

dysglycemia or dyslipidemia is newly identified, whether referral and treatment occur, and whether patients experience anxiety, stigma or unnecessary testing. Such studies could determine whether dermatology-based prompts add value in settings with limited access to primary care and which communication approaches support shared decision-making.

Research should also distinguish biologically and clinically different findings. Acanthosis nigricans, acrochordons, eruptive xanthomas, inflammatory dermatoses and androgen-related signs should not be combined into a generic "metabolic skin score" without evidence that such a score is valid. A modular approach may be more informative: one module for general cardiometabolic risk, another for urgent lipid-related presentations, and another for endocrine evaluation. Each would require independent derivation and validation.

Finally, studies should assess equity and implementation. Skin findings may be recognized differently across phototypes, and access to dermatology, laboratory testing and follow-up varies across populations. Digital photography or artificial intelligence may eventually support standardized assessment, but these tools would require careful evaluation for performance across skin tones, privacy and clinical workflow. The central research question remains whether adding skin-based information leads to better and fairer care than established risk assessment alone.

5. Conclusions

Acanthosis nigricans, multiple acrochordons and eruptive xanthomas can provide visible clues to metabolic abnormalities, but their diagnostic implications differ and depend on context. Hidradenitis suppurativa and psoriasis warrant attention to cardiometabolic comorbidity, while androgen-related findings should be evaluated through appropriate endocrine pathways. None of these signs alone establishes or excludes IR or metabolic syndrome.

When a relevant skin finding is observed, clinicians should confirm the diagnosis, assess overall cardiometabolic risk and use guideline-based laboratory testing. The skin can prompt earlier assessment, but biochemical criteria remain necessary for diagnosis.

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