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Dermal Glycation and Hyperpigmentation in Skin of Color with Type 2 Diabetes Glycation & Pigmentation in SOC with T2DM

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Abstract

Background: Advanced glycation end products (AGEs) are significant contributors to diabetes-related complications, causing systemic inflammation, vascular damage, and tissue aging. In the skin, chronic hyperglycemia leads to the nonenzymatic glycation of proteins, resulting in collagen cross-linking, loss of elasticity, and visible changes like stiffness, wrinkling, and hyperpigmentation. While these effects are well-studied in lighter skin types, their impact on individuals with skin of color (SOC) is less understood.

Objective: This study aims to examine ethnic variations in dermal glycation and the implications of AGE accumulation in SOC patients with type 2 diabetes mellitus (T2DM).

Methods: A narrative review of histological, biochemical, and clinical studies (2015–2025) related to AGE deposition, dermal matrix changes, and pigmentary abnormalities in SOC populations was performed. Relevant literature was sourced from PubMed, Scopus, and Embase.

Results: SOC patients display distinct dermal responses to glycation, including increased hyperpigmentation and changes in skin texture. AGE accumulation may be heightened in collagen-rich areas due to higher melanin and fibroblast activity in darker skin. Noninvasive AGE detection tools, like skin autofluorescence, may underestimate glycation in highly pigmented skin.

Conclusion: Understanding the differential effects of AGEs in SOC populations can improve early dermatological recognition of diabetes and support tailored management. Increased research inclusion of diverse skin types is needed to develop better diagnostic tools and targeted interventions for diabetic patients of color.

Keywords: Advanced Glycation End Products; Skin of Color; Type 2 Diabetes; Hyperpigmentation; Ethnic Disparities; Dermal Aging

Abbreviations: T2DM: Type 2 Diabetes Mellitus; AGE: Advanced Glycation End Products;

AN: Acanthosis Nigricans; SOC: Skin of Color; RAGE: Receptor for Advanced Glycation End Products; IL: Interleukin; TNF: Tumor Necrosis Factor; NF: Nuclear Factor; UV: Ultraviolet; PIH: Post-Inflammatory Hyperpigmentation; MCP: Monocyte Chemoattractant Protein; SAF: Skin Autofluorescence; CMYK: Cyan, Magenta, Yellow, Key; RCM: Reflectance Confocal Microscopy; DLQI: Dermatology Life Quality Index; AI: Artificial Intelligence; NAG: N-Acetylglucosamine; IGF: Insulin-like Growth Factor; IPL: Intense Pulsed Light; ALT: Alagebrium; PLLA: Poly-L-Lactic Acid; PASI: Psoriasis Area and Severity Index; SCORAD: Scoring Atopic Dermatitis; GLP: Glucagon-like Peptide;

Introduction

Type 2 diabetes (T2DM) leads to a variety of skin manifestations that mirror systemic metabolic problems. Chronic high blood sugar causes proteins and lipids to undergo nonenzymatic glycation through the Maillard reaction. This creates stable advanced glycation end products (AGEs) that build up in long-lasting tissues like skin [1]. Advanced glycation end products in the dermal layer cause collagen and elastin fibers to cross-link, leading to greater tissue stiffness, reduced elasticity, and tissue repair difficulties [1]. The clinical manifestation of AGE accumulation in diabetic patients includes premature aging features like thinning skin and wrinkles, a unique yellowish or sallow skin tone, and irregular pigmentation alterations [2]. Indeed, a “browning” phenomenon analogous to the caramelization of glycation in food products is thought to occur in vivo: Human diabetic skin contains a unique yellow-brown AGE pigment called AGE-Y which shows the direct role glycation plays in skin discoloration according to recent biochemical research [3-6] (Figure 1).

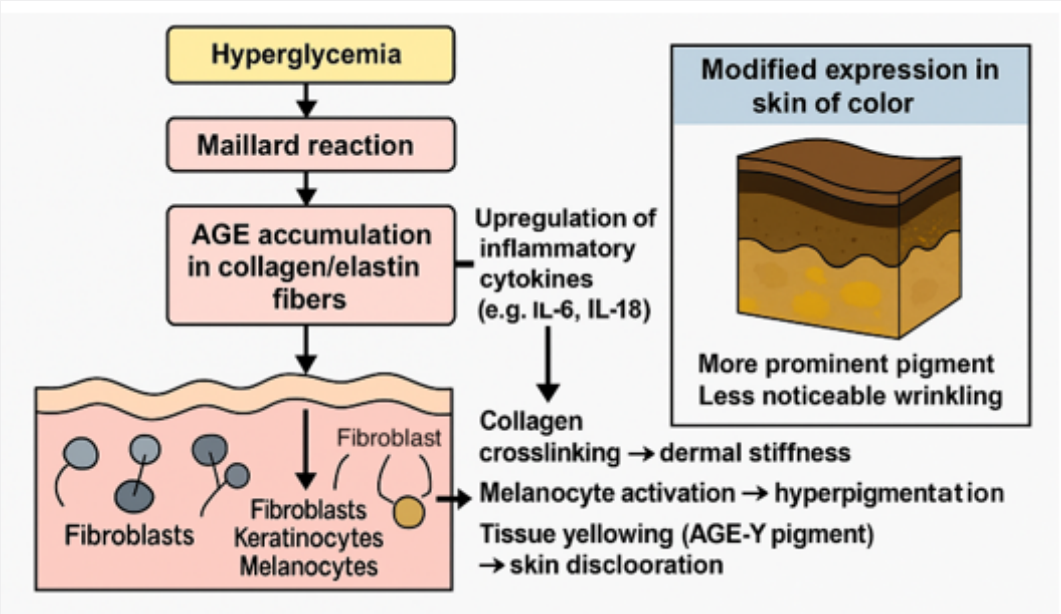

Figure 1: Mechanism of Dermal Glycation and Pigmentary Change in Diabetes.
This schematic illustrates the formation of advanced glycation end products (AGEs) through the Maillard reaction and their binding to RAGE on skin cells, leading to oxidative stress, cytokine release, and pigmentary changes such as post-inflammatory hyperpigmentation (PIH). The figure highlights collagen crosslinking, melanocyte stimulation via TNF-α and IL-18, and resulting changes in elasticity and pigmentation [1-3,5,6].

The skin manifestations observed in diabetes patients indicate deeper systemic health issues beyond cosmetic alterations. Insulin resistance and chronic hyperglycemia cause dermatologic conditions, including acanthosis nigricans (AN) and diabetic dermopathy. These skin changes sometimes appear before an official diabetes diagnosis and are early indicators of impaired glucose tolerance [7]. Recognizing these signs becomes essential when treating patients with skin of color (SOC) because they experience increased T2DM prevalence and risk factors, yet display dermatological manifestations that can present differently or receive insufficient attention. Individuals with higher Fitzpatrick phototypes ranging from III to VI belong to diverse ethnic groups such as African, Latin American, Asian, and Middle Eastern communities. They are collectively referred to as people of color. Populations with higher basal melanin content and unique skin structure experience modified glycation-related changes in their skin presentation. According to Glauner & von Stebut (2023), darker skin ages through uneven pigmentation and subtle surface changes instead of developing deep wrinkles, which occur in lighter skin [8]. The evidence indicates that dermal glycation in skin of color shows more signs of hyperpigmentation and dermal thickening than distinct rhytids, but research remains sparse.

Ethnic differences in diabetes outcomes underscore the importance of studying skin markers across all ethnic groups. Diabetes affects people of color at higher rates, and they face more severe complications from the disease. The dermatologic signs of diabetes, like rashes and pigmentation changes, often go unrecognized in SOC patients because of barriers to patients and healthcare providers [7,9]. The abundance of melanin in darker skin tones makes detecting erythema or subtle color shifts difficult, while providers frequently lack knowledge about diabetic dermatoses presentation in people with darker skin. The missed opportunities for metabolic disease detection through skin findings lead to delayed diagnosis and care management [7,9]. Increased recognition of glycation-related skin alterations in SOC populations would enhance both screening processes and culturally sensitive healthcare delivery. The performance of noninvasive diagnostic methods, such as skin fluorescence for AGEs, needs improvement in pigmented skin to prevent risk assessment gaps (Table 1) [10].

This narrative review explores how dermal glycation leads to hyperpigmentation in T2DM patients with skin of color. Our initial discussion addresses the biochemical processes through which AGEs form, their subsequent impact on skin architecture, and pigmentary control mechanisms. The discussion examines how ethnic and racial variations in skin biology, including melanin levels and dermal composition, impact these biological processes. An examination of how glycation-related dermal alterations present in skin of color will be conducted by focusing on common

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or distinctive hyperpigmented conditions such as AN, diabetic dermopathy, and post-inflammatory hyperpigmentation (PIH) in these populations. This study examines existing and novel diagnostic methods for skin AGEs and pigmentary disorders while identifying limitations for people with darker skin tones. The discussion concludes with therapeutic and preventive strategies to decrease AGE burden alongside safe hyperpigmentation management techniques for SOC patients. Integrating research findings from 2015 to 2025 will enable us to identify knowledge gaps and suggest research directions to enhance dermatologic and metabolic care for diabetic patients from diverse ethnic backgrounds.

Table 1: Key Differences in Diabetic Skin Presentation Between Light Skin and Skin of Color.

Feature	Light Skin (Fitzpatrick I-III)	Skin of Color (Fitzpatrick IV-VI)
Primary AGE Manifestations [8]	Wrinkling, thinning, yellowing	Hyperpigmentation, dermal thickening, uneven tone
Pigment Presentation [7]	Redness (erythema) more visible	Erythema may appear violaceous or undetectable
Common Skin Conditions [7,8]	Diabetic dermopathy (well-defined red/brown macules)	AN, PIH, lichenification
Diagnostic Challenges [9]	Subtle pigment changes easy to detect	Hyperpigmentation masks erythema; delayed recognition
Tool Performance (e.g., SAF reader) [10]	Generally accurate	Lower accuracy; melanin interferes with readings
Cultural Barriers to Care [10]	Often discussed openly	Stigma, fear of skin bleaching, misattribution (e.g., "dirt")

Abbreviations: AGE- Advanced Glycation End Products; AN- Acanthosis Nigricans; PIH- Post-Inflammatory Hyperpigmentation; SAF- Skin Autofluorescence.

Methods

A thorough literature review identified research relevant to glycation and hyperpigmentation, along with diabetes skin manifestations in skin of color between 2015 and 2025. The primary databases queried were PubMed, Embase, and Scopus, using combinations of keywords and Mesh terms, including: "advanced glycation end products," "glycation and skin," "diabetes AND skin hyperpigmentation," "skin of color," "ethnic skin AND diabetes," "skin autofluorescence," and specific condition terms such as "acanthosis nigricans," "diabetic dermopathy," and "post inflammatory hyperpigmentation." We conducted manual cross-referencing of bibliographies from relevant articles to collect additional references, which include essential background studies from before 2015 and foundational research.

The wide-reaching inclusion criteria represented the narrative (non-systematic) review approach. Our study selection included clinical research, such as randomized controlled trials and cohort studies, that investigated skin problems in people with diabetes, specifically when they reported results divided by ethnicity or skin tone. The study incorporated mechanistic and translational research to understand the effects of AGEs on skin cells through keratinocyte or fibroblast culture studies and animal models, while focusing closely on pigmentation pathways. General information from review articles and textbook chapters published since 2015 helped ensure comprehensive coverage of major topic areas, but data from original studies remained the focus. Our criteria permitted case reports because they demonstrated rare SOC presentations or new treatment methods. Research published in languages other than English was considered for inclusion when it contained an English abstract and its content met relevance criteria, such as Walha et al. 2024, which provided a French review about underdiagnosed diabetic skin lesions for contextual understanding of diagnostic difficulties [9].

The research team extracted data through qualitatively synthesizing selected publication findings instead of pooling quantitative results. Key points of interest included: The analysis focused on skin AGE accumulation variations between ethnicities, skin diagnostic device performance across skin tones, diabetic skin lesion descriptions in SOC, and intervention results for glycation or hyperpigmentation in diabetic patients. Research was narrative-based, which did not use formal quality assessments or bias scoring, but larger studies alongside analysis specific to SOC populations received increased consideration during conclusions formation. The results are presented in structured thematic areas below, which cover Pathophysiology, Ethnic Variations, Clinical Manifestations, Diagnostics, and Therapy to facilitate discussion clarity.

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Results

Pathophysiology of Dermal Glycation and Melanogenesis

Sustained high blood glucose levels in T2DM patients trigger the continuous production of advanced glycation end products within skin tissue, instigating structural alterations in the dermis and pigmentation shifts and during the Maillard reaction, reducing sugars bond with protein amino groups, which produce reversible Schiff base and Amadori products that transform into irreversible crosslinked moieties to form AGEs. The dermis experiences glycation primarily in its durable extracellular matrix proteins, including type I collagen and elastin. According to Lee et al. (2016), collagen fibers stiffen and become resistant to enzymatic breakdown when AGEs accumulate within them. The process of collagen crosslinking produces the stiffness and loss of elasticity seen in the skin of both diabetic individuals and older adults [1]. Glycation-induced crosslinking leads to reduced joint mobility that appears as diabetic characteropathy while also causing the distinct "waxy skin" characteristic of diabetic patients [7]. Reactive Di carbonyls produced during the glycation process generate oxidative stress, which damages proteins in the dermis and cell membranes, ultimately resulting in "Oxi glycation" or ox inflammation that creates chronic inflammation [11,12].

AGEs attach to cellular receptors including the receptor for advanced glycation end products (RAGE) which shows expression on multiple skin cell types such as keratinocytes, fibroblasts, endothelial cells, and melanocytes The AGE-RAGE interaction triggers intracellular signaling pathways that enhance proinflammatory cytokines and growth factors and increase matrix metalloproteinases leading to oxidative stress and extracellular matrix breakdown which contribute to chronic skin inflammation [5,13]. Interleukins (IL) and tumor necrosis factor-alpha (TNF-α) inflammatory mediators activate melanocytes, which boosts melanin production and leads to post-inflammatory hyperpigmentation, especially noticeable in darker skin types [13-15].

Recent scientific research has shown that AGEs affect melanogenic pathways within the skin. According to Lan et al. (2025), fibroblast activation through glycation results in melanocyte behavior changes mediated by cytokine release [5]. Furthermore, seminal work by Lee et al. (2016) discovered that melanocytes produce RAGE and that exposure to AGE-modified proteins boosts the activity of melanogenic markers. AGE exposure resulted in elevated expression of tyrosinase in human and murine melanocytes, which produced a 17–24% increase in melanin production above baseline. Effective suppression of melanin production occurred when researchers blocked the RAGE pathway using neutralizing antibodies or soluble decoy receptors, which proved that AGE-RAGE signaling directly stimulates melanocyte pigment production [1]. Extended AGE exposure for two weeks intensified melanin production, which validates the hypothesis that prolonged glycation stress leads to skin darkening. The link between poor glycemic control and skin hyperpigmentation becomes clear through these findings because sustained glucose exposure hastens skin aging while biochemically activating pigment production in melanocytes.

Dermal fibroblasts and the inflammasome function as key factors contributing to pigment formation through glycation and melanocyte activation via RAGE. The epidermal layer is affected by secreted factors from fibroblasts that have advanced glycation end-products due to diabetes or aging. Modern research demonstrates that the NLRP3 inflammasome is an inflammatory mediator during cellular functions [3]. Research by Fang et al. (2022) demonstrated that AGEs initiate NLRP3 inflammasomes in human dermal fibroblasts, leading to the release of IL-1β and IL-18 cytokines, which promote melanogenesis in nearby melanocytes [3,5]. The IL-18 binding protein blocked melanin and tyrosinase production in co-cultured melanocytes from AGE-treated fibroblasts, which indicates that IL-18 serves as a primary melanogenic signal in this setting [13]. Subsequent work by Lan et al. (2025) uncovered a sophisticated epigenetic pathway showing AGE-induced inhibition of anti-inflammatory protein A20 in fibroblasts through m6A-methylation of its mRNA, which results in degradation by the YTHDF2 reader protein. Without A20 protein activity, the inhibitory control over nuclear factor kappa beta (NF-kB) and NLRP3 inflammasome disappears, which leads to increased IL-18 secretion and greater melanocyte activity [5]. A direct link between fibroblast-mediated AGE-enhanced pathways and hyperpigmentation disorders was evidenced by increased dermal YTHDF2 together with reduced A20 that led to more epidermal melanin deposits in patients with chronic photodamage [5]. Glycation processes in the dermal layer lead to pigmentation changes via inflammatory signaling loops while simultaneously affecting melanocyte functionality.

Research studies need to understand how environmental factors affect glycation pathways alongside external variables. Ultraviolet (UV) radiation induces melanogenesis while at the same time it raises glycation levels. UV light exposure generates reactive oxygen species, accelerating AGE formation by initiating reactions between glucose and Di carbonyl compounds. UV light initiates a process where AGEs absorb radiation and emit fluorescence, which can result in harmful photodynamic cycles. A team led by Sultana et al. (2024) recently published their in vitro research study and revealed that skin cells experience more intense inflammatory responses and cellular aging when exposed to glyoxal and UVB irradiation than when exposed to each individually [13]. Individuals with diabetes who experience high sunlight exposure may develop skin aging and dyschromia faster. Internal hyperglycemia and external sources, such as charred and fried foods and smoking, create AGE accumulation that affects many T2DM patients. The skin deposits of exogenous AGEs lead to the dull yellowish complexion observed in some diabetic patients [2,4]. Accumulated AGEs, including AGE-Y chromophores,

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modify skin light reflection to create the diffuse dermal change known as "diabetic yellow skin" [3]. Scientific measurements of skin indicate that elevated skin AGE amounts produce greater yellowness in skin color (b value) and reduced brightness levels (L* value) [2]. For example, Zhang et al. (2025) found a strong correlation between skin AGE autofluorescence and facial skin yellowness, demonstrating how glycation affects skin complexion [2]. Together, these mechanisms illustrate a multifaceted picture: Dermal glycation results in structural skin changes and pigment alterations through RAGE signaling and inflammasome activation in response to environmental stressors that define the diabetic skin appearance.

Ethnic and Skin-Type Variations in Glycation Effects

Glycation happens throughout the body but affects skin appearance differently for various ethnicities and skin types. The unique structural and functional characteristics of the skin of color affect clinical manifestations of AGE deposition. These differences in glycation effects must be understood to fully comprehend why hyperpigmentation and other related changes occur more intensely or distinctly in skin of color patients with diabetes.

The distribution and content of melanin stand out as the main differences in skin of color. Fitzpatrick phototypes IV–VI individuals possess the same number of melanocytes as those with lighter skin but produce higher amounts of melanin, which is stored in larger melanosomes that remain separate instead of aggregating like in Type I-II skin. The plentiful melanin functions as a natural sunscreen by neutralizing reactive oxygen species and absorbing UV radiation, which helps protect dermal collagen against photoaging. The development of wrinkles and solar elastosis progresses more slowly in black skin compared to white skin that is the same age [8,16]. On the other hand, melanin can make pigmentary abnormalities both more visible and persistent. An inflammatory stimulus that produces a pale pink reaction in white skin could result in permanent brown spots on Black or Brown skin through post-inflammatory hyperpigmentation. The glycation process demonstrates that inflammation or microangiopathy outcomes caused by AGEs lead to more prominent hyperpigmented sequelae in skin of color [15,17]. The interaction between melanin and glycation processes may present unique biochemical interactions. Research shows melanin can attach to some metal ions and reactive carbonyls, which may control local glycation reactions, but it is not yet known whether this effect protects the skin or worsens it. While melanin may protect cells against glycation-induced oxidative stress because of its antioxidant properties [18], its presence in skin can lead to inaccurate autofluorescence measurements or undergo glycoxidation. Melanin's natural fluorescence and light absorption properties disrupt optical AGE measurements while diabetes-related melanin oxidation creates Melano lipofuscin, which adds to basal pigmentation [10,19].

Ethnic skin types show architectural differences in the dermis beyond differences in pigmentation. Research shows that the collagen fibers in black/African skin are more compact than those in white skin [16]. The dermal-epidermal junction has a threefold longer structure in African skin because it exhibits greater complexity with deeper rete ridges and dermal papillae than Caucasian skin. The pronounced interdigitation between layers contributes to mechanical strength and affects the diffusion of essential nutrients, oxygen, and metabolic byproducts such as AGEs through the epidermis and dermis. Researchers found that despite similar epidermal thickness, African skin exhibited increased papillary dermis convolution and fibroblast activity compared to European skin [20].

A study by Girardeau-Hubert et al. (2019) showed that papillary fibroblasts extracted from African-American donors generated increased amounts of growth factors such as keratinocyte growth factor and inflammatory chemokines like MCP-1 (Monocyte Chemoattractant Protein) compared to fibroblasts from Caucasian donors [21]. The hyperactive fibroblast behavior in SOC skin may lead to more intense dermal changes, such as remodeling and higher cytokine production, when AGEs build up in the dermis compared to lighter skin [22]. The presence of more numerous or larger fibroblasts in darker skin indicates an increased total amount of dermal protein available for glycation. Research shows that the dermis of black skin has more fibroblast nuclei and occasionally multinucleated giant fibroblasts, together with smaller collagen fiber bundles. Nevertheless, it maintains a larger overall collagen content. Research indicates that specific attributes in darker skin lead to distinct fibrotic conditions because of fibroblast and collagen variations, which cause a greater tendency for keloid formation [23]. Patients with diabetes may experience pronounced skin tightness or induration because their SOC contains increased collagen substrate and vigorous fibroblast activity, which leads to amplified Age-related cross-linking and dermal thickening.

The condition scleroderma diabetic rum manifests as woody hardening of the upper back and neck in patients with longstanding type 2 diabetes mellitus and serves as a clinical example of differential glycation changes. Researchers found that case series indicate more frequent diagnoses of this condition in lighter-skinned individuals because signs like erythema and peau d'orange texture stand out more clearly. Skin of color (SOC) patients experience delayed diagnosis of dermal changes due to less visible clinical signs until the condition advances. Dermal glycosaminoglycan buildup and collagen glycation initiate skin thickening and reduced elasticity. SOC patients experience disease progression through less noticeable visual indicators, which leads to diagnostic delays as thick skin may be misconstrued as normal ethnic skin variation instead of a pathological issue [15,24]. The brown atrophic macules known as diabetic dermopathy or "shin spots" on the shins result from minor dermal ischemia combined

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with collagen glycation, marking diabetic microangiopathy. These lesions can become indistinguishable from natural skin coloration in individuals with deep skin tones or appear as trauma-related pigmentation changes, which leads to their neglect during routine examinations. Research shows that diabetic dermopathy affects nearly 30% of diabetic patients in general [25,26], yet studies about its occurrence in patients with skin of color remain limited. The prevalence of diabetic dermopathy remains consistent across different skin tones. Yet, documentation of this condition is less common among people with skin of color because of diagnostic bias or the difficulty of seeing subtle pigmentation shifts on darker skin. This underscores the importance of heightened clinical suspicion: Any new dark brown lesion appearing on the pretibial area of a patient with diabetes should prompt consideration of diabetic dermopathy, regardless of the patient's skin tone, because it could indicate microvascular dysfunction [15,25].

Skin barrier function and immune cell distribution show significant differences between skin types. Research indicates that the stratum corneum of black skin exhibits a slightly lower pH and higher lipid concentration compared to white skin, while containing more inflammation-prone features such as enlarged mast cell granules and greater tryptase content [16,23]. Researchers have noted an increased number of dermal macrophages present in darker skin types [23]. The inflammatory environment may be affected by these factors during glycation processes. When AGEs bind to RAGE on immune cells such as macrophages and mast cells, they release inflammatory mediators, including TNF and IL-6. People with more or hyper-responsive mast cells in their skin may experience stronger edema and pigmentary changes because mast cell mediators can influence melanocyte behavior. According to Glauner & von Stebut (2023), individuals may experience more severe post-inflammatory hyperpigmentation following an insult in SOC because of this intense inflammatory reaction [8]. The presence of melanin could lessen oxidative stress, which leads to glycation and potentially prevent the formation of AGEs from occurring initially. Research has yet to fully outline how these contradictory elements result in a definitive outcome.

The reliability of noninvasive skin AGE measurements varies based on skin pigmentation. Ultraviolet light excitation-based Skin Autofluorescence (SAF) assessment devices have received validation mostly in European populations as indicators of total tissue AGE load. Melanin exhibits fluorescence and, most importantly, absorbs excitation wavelengths intensely, which leads to decreased AGE signal detection in people with darker skin tones [10,19]. A study by Ahdi et al. (2015) discovered that African and South Asian diabetic patients had lower SAF values than Dutch diabetic patients with comparable diabetes durations, regardless of similar skin color intensity. A large number of dark-skinned subjects were removed from the analysis because they exhibited very low reflectance (R%), which led to unreliable device penetration and fluorescence interpretation. Skin AF maintained its correlation with complication status for lighter skin yet demonstrated weaker predictive accuracy for darker skin after excluding low-reflectance subjects. SAF assessment proves helpful for evaluating complication risks in patients with light skin; however, its effectiveness declines for darker skin, even after removing low-reflectance subjects [19]. Current AGE readers might fail to detect adequate glycation levels in patients of color, resulting in misleadingly low risk assessments for cardiometabolic issues. Current research endeavors focus on modifying device algorithms to account for skin pigmentation differences and explore alternative wavelengths to minimize melanin absorption [19,27]. Clinicians must approach SAF results deemed "normal" for Black or deeply pigmented patients with careful interpretation until these technologies receive validation. The existing technical problem demonstrates the essential need for research that includes all skin types to adjust diagnostic limits and determine if SOC naturally gathers AGEs differently or if this is a measurement artifact.

Ethnic differences in skin characteristics, ranging from microscopic features like fibroblast density to macroscopic traits such as pigmentation and thickness, affect how dermal glycation manifests and is detected. Although SOC patients exhibit the same or higher levels of AGE deposition when compared directly, their skin shows most prominently pigmentary changes and increased dermal firmness instead of severe wrinkling. The variability among diabetic dermatoses highlights why a standardized approach should not be used for all patients. Upcoming sections will demonstrate how the detailed pathophysiologic differences result in observable clinical hyperpigmentation patterns in diabetic patients with darker skin tones and describe the diagnostic and treatment challenges these conditions present.

Clinical Manifestations of Glycation-Associated Hyperpigmentation in Skin of Color

Individuals with skin of color who have T2DM frequently display hyperpigmentation patterns, including diffuse and localized areas that correlate with glycemic status, insulin resistance, or tissue alterations caused by glycation. Although these skin findings are not unique to SOC, they require special attention in darker skin due to factors like severity, diagnostic delays, and cosmetic consequences. This paper presents the primary hyperpigmented lesions and skin changes found in diabetic patients while highlighting their manifestations in individuals with skin of color.

Insulin resistance and T2DM frequently present on the skin as acanthosis nigricans, one of the most prevalent dermatological signs. AN manifest through velvety hyperpigmented plaques that show gray-brown to black coloration and are usually located in skin fold areas such as the neck, axillae, groin, as well as inner elbows, knuckles, and even the forehead [28]. Clinicians sometimes mistake mild AN for harmless skin differences or leftover dirt. However, AN in diabetic individuals is typically pathognomonic: The disease presents with symmetric distribution and a unique

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skin texture characterized by thickened velvety layers and a slightly papillomatous appearance [29]. A cross-sectional study demonstrated that AN affected 19% of diabetic patients, while those with AN had double the odds of having T2DM compared to those without the condition [25]. Notably, AN appears to affect people with darker skin disproportionately: Research data show that Black, Hispanic, and Native American patient groups generally have increased instances of obesity-related AN [7,25]. The review shows published Cases mostly feature Fitzpatrick skin types III-V, but precise epidemiologic data by race remain limited.

AN pathogenesis shows a direct connection to hyperinsulinemia instead of glycation processes. Insulin levels rise in T2DM and metabolic syndrome patients, which activate keratinocytes and fibroblasts through insulin-like growth factor receptors, resulting in epidermal cell multiplication and fibroplasia in dermal layers. The affected skin experiences increased melanin deposition because the cells release melanocyte-stimulating cytokines. Insulin is a skin growth factor, leading to excessive growth faster than normal skin shedding and accumulating thickened, dark skin. The natural melanin levels in skin of color intensify AN's appearance, and persistent rubbing in thickened skin areas due to acanthosis leads to melanosis. The posterior neck is a typical location where AN show pronounced characteristics that often lead to misdiagnosis as poor personal hygiene. Research conducted by Dhanoo et al. (2024) led to the development of innovative diagnostic methods. The research group created a cam smartphone application that evaluates neck acanthosis color intensity to estimate insulin resistance and demonstrated robust performance across different skin tones as a screening tool. The application examined the black (K) channel of the CMYK (Cyan, Magenta, Yellow, Key) color space, which showed the highest correlation with hyperpigmentation levels and successfully identified diabetic or prediabetic conditions with high sensitivity [29]. This tool stands out for SOC because it objectively measures pigment instead of depending on clinician assessment, which helps minimize subjective bias. Management of acanthosis nigricans requires patients to focus on weight reduction and insulin sensitivity enhancement, which physicians typically achieve through medications like metformin. The interventions documented by Ly et al. (2025) and Abate et al. (2025) produce gradual pigmentation and skin texture enhancements. Persistent cosmetic conditions have been treated through topical options like retinoids, vitamin D analogues, and procedural approaches, including mild chemical peels and laser therapy [23,25]. Patients with skin of color must approach treatment with caution in order to prevent the development of post-inflammatory hyperpigmentation. When these treatments are conducted using proper technique and appropriate patient selection, they can be successful while minimizing adverse pigmentary effects [17,30]. A function as a significant marker of metabolic health in SOC patients while presenting as a cosmetic issue that intersects with cultural beliefs (patients commonly apply lightening creams to their necks and steer clear of specific garments). The detection of this condition initiates a need for diabetes screening and related disorders because skin manifestations often reveal systemic diseases [7].

Diabetic Dermopathy manifests as small, round or oval brown spots located on the shins in the pretibial areas. The lesions known as "shin spots" develop from minor blood leakage followed by hemosiderin and melanin deposition within the dermal layer due to diabetic microangiopathy and neuropathy [7,25]. Initially presenting as slightly scaly red or purple papules, these lesions transform into atrophic hyperpigmented macules, which remain visible for many months. Light skin displays these lesions as distinct red-brown patches. In contrast, dark skin shows them as different brown or gray shades, which might be mistaken for post-traumatic marks or misidentified as tinea infections or lichen planus pigmentosus without careful examination. Dermoscopy reveals a brown background with faint erythema, which may not be visible to the naked eye in SOC. Research shows that diabetic dermopathy affects approximately 20-50% of diabetic patients and becomes more common as both age and diabetes duration increase, according to various studies. These lesions are harmless and do not need treatment because they usually fade away after 1-2 years, while new lesions continue to develop [25]. However, the clinical significance of diabetic dermopathy lies in its strong association with systemic complications: Multiple shin spots appear to raise the likelihood of developing diabetic retinopathy as well as nephropathy and neuropathy, according to research by Ly et al. (2025) and Natarajan (2024) [25,31]. Immediate identification of all skin types remains a fundamental requirement. The reduced color contrast in skin of color patients necessitates a higher suspicion level. Healthcare providers should examine the anterior shin through touch because the atrophic texture of dermopathy remains detectable even when skin pigmentation alterations are not visually apparent. A skin biopsy becomes necessary when diagnosis remains uncertain because it shows dermal melanin deposition along with hemosiderin and extravasated red blood cells, while also excluding potential mimickers like cutaneous amyloidosis and leukocytoclastic vasculitis [15]. The appearance of diabetic dermopathy should lead doctors to assess glycemic control and screen for microvascular complications according to [25]. Patients with SOC who display these lesions and have no known history of diabetes should undergo blood glucose testing alongside HbA1c measurement and possibly an eye examination. Physicians need to proactively check the skin of diabetic patients since dermopathy remains unnoticed, mainly by patients, due to its asymptomatic nature and small size.

Special dermatologic care is essential for diabetic patients with skin of color because their condition leads to post-inflammatory hyperpigmentation. Diabetes patients experience skin damage, including infections and ulcers, which often result in permanent hyperpigmented marks or scars even after healing.

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Skin of color patients experience longer-lasting and more intense pigmentary sequelae because their melanin activity is increased while their wound healing process is delayed. Dark skin patches often remain after healing from diabetic foot ulcers. In contrast, eczematous and psoriatic skin issues worsened by high blood sugar levels and candidal infections in skin folds, like under the breasts, leave behind lasting brown marks. Post-inflammatory hyperpigmentation in scar tissues remains for months to years without proactive management.

Diabetic neuropathy worsens this problem since patients cannot detect minor injuries, which leads to slow wound healing because of glycation and microvascular damage, which raises the chances of skin pigmentation from hemosiderin or melanin buildup [24]. The formation of "pigmented pretibial patches" represents an underreported yet significant glycation-related phenomenon that displays clinical similarities to diabetic dermopathy [15].

Rubeosis faciei diabetica represents another pigmentary change in diabetes that manifests as facial erythema because of capillary dilation. Intensely pigmented patients may show a diffuse brown or ashen appearance across their cheeks in place of redness, which can be confused with melasma or sun-induced hyperpigmentation unless metabolic conditions of the patient are evaluated [10,17]. Identifying the specific causes of facial hyperpigmentation in SOC, like melasma, PIH from acne or eczema, and the "diabetes mask," presents significant diagnostic challenges. Wood's lamp examination helps determine pigment location between the epidermis and dermis. At the same time, reflectance confocal microscopy reveals basal layer hyperpigmentation and dermal Melano phages, both of which assist clinical judgment and treatment planning [10].

The study by Pagan et al. (2022) explains how dermatoscopic patterns and Reflectance Confocal Microscopy (RCM) mosaics help identify hyperpigmentation disorders in skin of color. PIH reveals a dermal blue-gray color through dermoscopy, while melasma demonstrates a fine brown reticulated pattern. Such distinctions are important because treatment differs: Top-layer pigmentation improves with creams and peels, while deeper dermal pigmentation from long-term glycation-related PIH often needs laser treatment or shows remarkable resistance [10].

Medical literature reports generalized skin darkening in some diabetes patients based on anecdotal evidence but lacks sufficient quantification and comprehensive reporting. Medical professionals report that those with uncontrolled diabetes often show skin that becomes increasingly dull and dark over time. The observed skin discoloration results either from yellow-brown glycation pigments like advanced lipofuscin-like compounds mixing with native melanin or from the cumulative pigmentary alterations caused by AN and post-inflammatory hyperpigmentation across several body sites. Patients with skin of color may not notice these subtle changes unless healthcare providers compare darker dorsal skin with lighter palms or analyze older photographs. The findings emphasize that metabolic dysfunction indicators in the skin can spread widely and develop slowly while remaining unnoticed in individuals with skin of color unless screenings are thorough. Recent findings demonstrate that AGEs and their derivatives build up in skin tissue and affect pigmentation pathways through their interaction with melanin and glycation byproducts in the presence of oxidative stress, leading to gradual skin darkening [2,6].

A research investigation conducted on an Indian cohort by Karra et al. (2024) discovered that widespread hyperpigmentation patterns were more common among patients with elevated HbA1c levels. This study observed that patients with higher HbA1c levels showed more frequent overall hyperpigmentation, such as diffuse facial pigment or knuckle darkening, which indicates that poor glycemic control may lead to generalized pigmentation. Despite numerous confounders such as obesity and genetic factors, chronic glycemia may affect baseline melanogenic tone [26]. The persistent low-grade inflammation associated with diabetes may lead to a subtle increase in melanocyte activity throughout the body.

Necrobiosis lipoidica, a diabetic skin condition, affects SOC individuals by manifesting with violaceous or brown borders that differ from the classic red-orange rim observed in lighter skin types [32]. When the yellow papules from eruptive xanthomas resolve, they leave behind post-inflammatory hyperpigmentation following sudden triglyceride level increases [23]. Some diabetic patients exhibit perifollicular darkening on their shins and forearms, which may be caused by glycation of follicular basement membranes, leading to pigment incontinence [24]. These relatively minor or uncommon skin manifestations indicate that health practitioners should perform thorough and culturally sensitive skin examinations for diabetic patients.

Patients of color with diabetes often show hyperpigmented skin conditions that routine examinations miss frequently. Both AN and diabetic dermopathy are key indicators for performing a metabolic assessment. Skin injuries in diabetic patients often result in significant and persistent PIH within SOC, which adds to dyschromia burdens. Quality of life suffers when uneven skin tone and unsightly dark patches create distress and impact psychosocial well-being [28]. Patients from different cultural backgrounds may explain these skin changes as natural aging or inherent racial traits, leading them to avoid medical consultation or seek hazardous bleaching solutions. Medical professionals must inform patients that these skin changes occur during the disease process but can show improvement through correct diabetes management. Clinicians who treat pigmentary changes as valuable clinical information instead of cosmetic problems can enhance metabolic outcomes through root cause treatment and patient skin management with safe dermatologic therapies. This

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section explains the diagnostic tools and methods for improving SOC conditions while addressing their limitations.

Diagnostic Tools and Challenges in Skin of Color

Thorough clinical examination remains key for diagnosing skin manifestations of diabetes in SOC patients, while adjunctive tools offer additional benefits for detection and monitoring. The diagnostic tools mentioned encounter specific problems when used on patients with darker skin tones. This section presents established and novel diagnostic methods to assess dermal glycation and hyperpigmentation specifically for skin of color.

Clinical examination and grading

In diabetes patients with skin of color, dermatologic assessment depends primarily on clinical examination and grading. Standardized high-resolution photography combined with a detailed visual examination provides essential documentation of baseline pigmentation and enables monitoring of changes over time or after treatment. Consistent lighting conditions and cross-polarized filters reveal subtle pigmentary transitions by reducing surface glare, which improves visualization. Polarized images provide clearer visibility of both the AN patch borders and the underlying erythema found in early necrobiosis lipoidica [33].

Clinicians use objective assessment tools to evaluate pigmentation severity, including color charts and validated reference photos such as those provided by the Dermatology Life Quality Index (DLQI). The ANcam smartphone application represents a new digital innovation that shows promise for advancements in this field. The device measures skin pigmentation of the neck area and strongly agrees with clinical insulin resistance evaluations. In their validation study, Dhanoo et al. (2024) confirmed that the app's color grading closely matched endocrinologist assessments and provided an affordable, non-invasive screening option suited for communities of color where laboratory testing access is limited [29].

Ultimately, heightened clinical vigilance is paramount. Healthcare professionals must actively inspect frequent dermatologic change locations, including the neck, axillae, shins, knuckles, oral mucosa, and feet, during each diabetes patient visit. SOC patients require special attention because pigmentary changes often become subtle or blend into the natural skin tone.

Wood's Lamp (UV light)

The Wood's lamp examination, which utilizes UV light, is an inexpensive instrument within clinical settings to distinguish between pigmentation found in the epidermis and that in the dermis. Exposure to long-wave ultraviolet light causes epidermal melanin to darken while dermal pigmentation stays the same because UV rays do not penetrate beyond the basal layer. A Wood's lamp assessment helps determine if facial pigmentation is epidermal, such as melasma or PIH, which respond to treatments, or dermal, indicating more persistent conditions [10].

Deeply pigmented skin reduces the effectiveness of Wood's lamp examinations because the high levels of melanin decrease contrast, which makes findings difficult to discern. While high melanin levels may limit its effectiveness, the device remains useful for assessing localized lesions when combined with clinical context and visual asymmetry evaluation. For individuals with skin of color who display subtle and widespread hyperpigmentation conditions, clinicians must use tools such as the Wood's lamp to enhance their clinical assessments of pigmentation levels and determine proper treatment methods [17].

Dermoscopy

Skin surface microscopy with magnification and polarized light technology, known as dermoscopy, is an essential noninvasive diagnostic instrument for pigmentary disorder assessment, particularly in skin of color. Distinct patterns revealed through dermoscopy help in the differential diagnosis of hyperpigmented lesions. AN reveal a characteristic "skin furrow" pattern with parallel ridges due to underlying papillomatosis, whereas diabetic dermopathy presents a fine reticulated pigmentation pattern with poorly defined borders. In PIH cases, medical professionals may sometimes identify granular gray-brown dots that represent dermal Melano phages, which indicate pigment depth and chronicity [15,24].

A recent review pointed out the importance of dermoscopy for SOC analysis because pigmentary disorders such as melasma, PIH, and exogenous ochronosis often present overlapping clinical features. Melasma manifests with a pseudo-network pattern of fine light brown pigmentation while keeping follicular openings intact, yet PIH and ochronosis display more irregular blotchy pigmented clusters [10]. Dermoscopy helps differentiate between dermopathy and conditions like stasis purpura or early vasculitis lesions in diabetic patients by identifying superficial scaling and a consistent pigment network within hyperpigmented shin patches. Noninvasive dermoscopy has become a natural part of outpatient dermatology and primary care procedures while improving diagnostic precision across varied patient groups.

Skin Autofluorescence (AGE Reader)

The Skin Autofluorescence (AGE Reader) technique enables researchers to noninvasively measure advanced glycation end products on the skin by detecting specific fluorophores like pentosidine. Devices called AGE Readers emit ultraviolet light around 370 nm wavelength onto the inner forearm skin to measure emitted fluorescence, which translates into a numeric SAF score linked to long-term high blood sugar and potential diabetes complications [26,32]. Research has integrated SAF scores into risk assessment models for microvascular and cardiovascular diseases among populations with lighter skin tones.

The accuracy of SAF assessments proves challenging when applied to individuals with darker skin tones. The intense UV excitation absorption and broad-spectrum fluorescence emission

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from melanin disrupts AGE-specific signals. Melanin absorption of UV light reduces SAF readings, which results in underestimated glycation burden measurements for skin of color [19,27]. Early validation studies presented initial methods for skin tone adjustment through reflection indices. Initial validation studies that applied reflection indices demonstrated significant variability when measuring intensely pigmented subjects. Notably, Ahdi et al. (2015) removed about one-third of Black African participants because their skin reflectance was inadequate. They found non-European groups had lower SAF scores despite equal HbA1c levels, which showed a calibration bias [19].

Recent studies demonstrate that relative SAF levels in ethnic subgroups remain linked to diabetes-related complications even when absolute measurements vary between races or skin tones because higher SAF readings in Black individuals still signify greater personal risk compared to others with similar pigmentation [27]. The SAF score of 2.5 in a Black patient does not hold the same meaning when compared with a White patient having the same score.

Upcoming devices will achieve better accuracy by using different excitation wavelengths, like visible light, or by embedding skin tone algorithms into calibration models [3]. Clinicians need to record the Fitzpatrick skin type during SAF assessments in SOC and carefully evaluate borderline results until better methods become available. Clinicians should not mistakenly feel reassured by a "low" SAF reading when treating patients with longstanding uncontrolled diabetes who show visible glycation signs or other risk factors. When necessary, situations arise, healthcare professionals must consider additional tests like conventional HbA1c levels, complication screenings, and serum or urinary AGE assays in research settings to precisely evaluate metabolic burden.

Reflectance Confocal Microscopy (RCM)

RCM represents a noninvasive imaging method that applies near-infrared laser technology to achieve nearly histologic resolution visualization of epidermal and superficial dermal structures within living tissue. Traditionally, this technique has been used for dermatologic research and specialized clinical work to evaluate pigmented lesions such as melanoma versus benign nevi and to study pigmentary disorders, including melasma and post-inflammatory hyperpigmentation. RCM proves especially beneficial for skin of color affected by hyperpigmentation since it avoids the melanin absorption limitations present in ultraviolet-dependent devices such as Wood's lamp or AGE Readers. RCM produces reflectance-based images that enable high-resolution assessments of skin conditions without being influenced by pigmentation [10].

RCM identifies bright granular particles within the dermis during dermal PIH examination that match melanin-rich macrophages (Melano phages), thus validating pigment incontinence. The tool enables visualization of epidermal thickening and both basal cell hyperpigmentation and rete ridge flattening, which may correlate with chronic glycation or photoaging and overuse of topical corticosteroids [33].

Although RCM cannot detect AGEs directly, emerging optical technologies such as multiphoton microscopy have been tested for this application. Pentosidine AGEs demonstrate autofluorescence when multiphoton excitation is used, which suggests that new imaging platforms might eventually allow noninvasive assessment of skin glycation levels through an "optical skin biopsy" approach [2,3]. Despite being experimental at this stage, these techniques hold potential for future evaluation of structural and biochemical changes in diabetic skin, particularly in SOC patients who require more reliable assessment methods.

Biopsy and histopathology

The definitive method for diagnosing uncertain skin conditions and directly assessing glycation-related tissue changes relies on biopsy and histopathology. Histological differentiation of pigment sources and structural alterations through skin biopsies provides essential insights in complex cases and research applications. Immunohistochemical staining with antibodies specific to AGEs, such as anti-carboxymethyl-lysine or anti-pentosidine, enables researchers to visualize glycation deposits in the dermal layer. Diabetic patients' skin biopsies display AGE deposits forming a rim-like pattern around dermal blood vessels and within the papillary dermis, which indicates microvascular damage [3,6]. Research through comparative histology demonstrates that sun-exposed skin has significantly greater AGE accumulation than photo protected skin, which implies UV radiation speeds up dermal glycation processes [1].

Histologic stains provide information about the location of pigmentation and the underlying cause in pigmentary disorders. Melanin detection happens through Fontana-Masson staining, and Perl's stain verifies iron deposits when hemosiderin is suspected. Histological examination of diabetic dermopathy usually shows melanin incontinence wherein melanin enters the dermis together with hemosiderin deposits that result from erythrocyte leakage, thus validating dual pigmentary processes [7]. The distinctive features of papillomatosis and hyperkeratosis with mild basal layer hyperpigmentation observed in acanthosis nigricans biopsies serve to separate it from similar conditions such as confluent and reticulated papillomatosis and dermatosis papulose nigra [32].

Biopsy procedures are usually unnecessary for typical presentations but become essential when dealing with atypical cases, especially in patients of color, because overlapping pigmentary patterns and natural melanin levels could hide diagnostic signs. Histopathological examination becomes essential for differentiating suspected malignant (paraneoplastic) AN with its rapid onset and mucosal involvement from benign diabetic AN. The natural darkness of SOC patients' skin tones makes dramatic clinical changes hard to detect until they progress significantly, underscoring the need for early biopsy when symptoms appear worrisome [25].

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A multimodal strategy is essential for accurate dermatologic assessment in SOC. Traditional diagnostic tools, including color-based photography and Wood's lamp, face limitations when applied to darker skin because of melanin interference. A meticulous, complete-body visual and tactile assessment continues to be the basic approach. Dermoscopy, alongside standardized photography and Wood's lamp examination, proves beneficial for examining localized lesions. When clinical uncertainty continues, doctors perform a biopsy as the decisive step.

We expect new diagnostic tools to become more inclusive of ethnic diversity as technology progresses. Real-time melanin quantification will enable future AGE readers to adjust fluorescence interpretation, and machine learning applications trained with diverse image datasets will enhance tele dermatology accuracy. Initial Artificial Intelligence (AI) models demonstrate potential in identifying nail fold capillary irregularities in diabetes patients, which could lead to the photographic detection of AN or dermopathy [2]. We must include SOC in datasets to prevent diagnostic bias and achieve fair dermatologic treatment for every patient.

Therapeutic and Preventive Implications

To effectively handle the skin complications arising from glycation and hyperpigmentation in diabetic patients, healthcare providers must implement an integrated approach that targets both general metabolic disease control and specific skin treatment methods. The main objectives of patient care involve enhancing quality of life by lessening visible distressing pigment changes while reducing health risks due to predictive cutaneous systemic complications and ideally reducing or reversing skin damage caused by advanced glycation end products. Treatment plans for hyperpigmentation include glycemic control optimization and lifestyle changes, as well as specific topical and procedural interventions. Researchers are studying new systemic and topical anti-glycation treatments to determine their effectiveness in stopping dermal glycation progression. The safety, efficacy, and aesthetic aspects of skin of color require careful consideration because pigmentary disorders frequently remain persistent and carry significant psychosocial burdens [25].

Optimal Glycemic Control

While this may appear obvious, maintaining strict glycemic control remains essential for preventing glycation-related skin damage associated with diabetes. Research conducted over time proves that continuous management of glucose levels leads to substantial reductions in tissue AGE buildup. Patients with type 1 diabetes who maintained intensive control of their blood sugar levels showed lower levels of skin collagen AGEs according to SAF measurements and demonstrated fewer systemic complications [27]. Minor improvements in HbA1c benefit patients with type 2 diabetes because each 1% reduction leads to decreased AGE accumulation according to Staubach & Staubach (2024), as chronic high blood sugar causes proteins to undergo nonenzymatic glycation [24].

Visible skin improvements typically occur when patients achieve better glycemic control. Patients observe partial regression of acanthosis nigricans through declining insulin levels while experiencing faster wound healing and reduced post-inflammatory hyperpigmentation. Understanding how blood sugar regulation safeguards vital organs such as the eyes and kidneys while leading to improved skin health serves as powerful motivation for patients, particularly in communities where skin appearance heavily influences psychological and social well-being. For example, a provider might say: Good blood sugar management can cause the dark skin patch on the neck to disappear gradually. Thus, improvement of dermatologic conditions gives patients a clear and motivating sign of systemic health progress, enabling providers to engage them using visual signs that resonate culturally.

Dietary Modifications

Adjustments to diet can be an important aid for diabetic patients to lessen glycation effects on their skin. Reducing the consumption of external AGEs in grilled, fried, and baked foods may lower overall glycation levels within the body and skin. Cooking methods like grilling and frying produce preformed AGEs, including carboxymethyl-lysine and methylglyoxal derivatives that build up in body tissues such as skin [6]. Clinical research has shown that people who consume a diet with minimal AGEs through cooking methods such as steaming or boiling experience lower levels of circulating AGEs and oxidative stress markers. However, there is limited direct evidence connecting dietary AGE reduction to skin-specific health benefits [24].

Antioxidant-rich foods such as fruits and vegetables and flavonoid intake may reduce glycation through the neutralization of reactive carbonyl species. Research shows that flavonoids in cocoa, green tea, and selected berries capture glycation intermediates while decreasing oxidative damage. Research models demonstrate the antiglycation capabilities of these compounds, as Zhang et al. (2025) discovered that dietary polyphenols and AGE inhibitors affect skin aging parameters and enhance dermal resilience. According to the study, dietary polyphenols alongside AGE inhibitors can alter skin aging metrics while enhancing skin resilience [2].

A small clinical study discovered that oral carnosine treatment reduced SAF in type 2 diabetes patients over a multi-month period [3]. As it becomes more integrated into mainstream healthcare practice, the strategy of recommending patients to "eat for your skin"-which focuses on a diet low in glycemic index and high in antioxidants while minimizing processing-offers significant benefits with minimal risk. The nutritional guidance supports existing diabetes management plans while promoting patient participation through visible skin health enhancements.

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Topical Antiglycation and Pigment-Modulating Agents

The cosmetic industry's focus on reducing visible skin aging led to the recent development of topical antiglycation agents for treating glycation-induced skin changes and hyperpigmentation. Various active components demonstrate potential benefits for diabetic patients with skin of color who experience lasting pigmentary changes that affect psychosocial well-being (Table 2).

Table 2: Topical Agents for Glycation and Hyperpigmentation in Diabetic Skin.

Agent	Mechanism of Action	Evidence in Diabetes	Notes for SOC Use
Aminoguanidine [6]	Binds reactive carbonyls; inhibits AGE formation	Preclinical UV-skin models; no clinical data	Potential topical benefit; lacks human studies
Carnosine [12,13]	Antioxidant, chelates metals, scavenges aldehydes	Low carnosine in diabetic skin; improves elasticity	Non-irritating; used in cosmeceuticals
Niacinamide + NAG [34]	Reduces melanosome transfer and tyrosinase glycosylation	Improves facial hyperpigmentation in trials	Well-tolerated; barrier-repairing benefits
Thiamidol [17]	Tyrosinase inhibitor; reduces melanin synthesis	Effective in SOC for melasma and PIH	Minimal irritation; suitable for shins and neck
Glutathione [35]	Antioxidant; shifts melanogenesis to pheomelanin	Small study showed hyperpigmentation reduction	Controversial use; oral route safer than injections
Azelaic Acid [32]	Tyrosinase inhibition; anti-inflammatory and anti-IGF effects	Used off-label for PIH and mild AN	Safe alternative to hydroquinone; less risk of PIH

Abbreviations: AGE- Advanced Glycation End Products; UV- Ultraviolet; NAG- N-Acetylglucosamine; PIH- Post-Inflammatory Hyperpigmentation; AN- Acanthosis Nigricans; IGF- Insulin-like Growth Factor; SOC- Skin of Color.

Aminoguanidine, which was studied early for inhibiting glycation, bears the alternative name pimagedine. The systemic application of this compound in diabetes treatment ceased because of harmful effects, but researchers have examined its use in topical skin treatments. According to Kabita et al. (2024), aminoguanidine creams proved effective in reducing AGE buildup through reactive carbonyl intermediate binding in UV-exposed skin models [6]. Yet, human clinical trials on this treatment remain scarce. The naturally occurring dipeptide carnosine (β-alanyl-L-histidine) demonstrates properties that protect against glycation while also acting as an antioxidant. This compound binds metal ions while simultaneously neutralizing the aldehydes participating in AGE formation. Scientific studies reveal decreased carnosine concentration in diabetic skin, which proposes possible advantages through topical supplementation [12,13]. Carnosine-based cosmeceuticals have been shown to improve skin firmness and elasticity because they likely inhibit collagen crosslinking.

Niacinamide (vitamin B3) and N-acetylglucosamine (NAG) have established reputations for modulating skin pigmentation rather than acting as direct antiglycation agents. Niacinamide enhances the skin barrier function while reducing melanosome transfer, and NAG blocks tyrosinase glycosylation. Clinical research showed that facial hyperpigmentation diminished after eight weeks of using a moisturizer with niacinamide and NAG ingredients [34]. These agents can reduce inflammation and improve the skin barrier in diabetes patients.

Thiamidol (isobutylamido thiazolyl resorcinol) represents a novel non-hydroquinone tyrosinase inhibitor tested exclusively for SOC patients. A 2024 research study by Schuster and Sammain showed that topical thiamidol treatment led to substantial melasma and PIH improvements in Fitzpatrick skin types IV-VI while producing minimal irritation, thus proving to be an effective treatment for diabetic hyperpigmentation in cosmetically sensitive areas like the neck or shins [17].

The powerful antioxidant glutathione has become a widely depigmenting treatment throughout Asian market. In diabetic patients, Soban et al. (2021) indicate that taking glutathione by mouth can help decrease widespread hyperpigmentation. The modulatory effect of this substance on melanogenesis involves the transition from eumelanin to pheomelanin production while also detoxifying oxidative intermediates [35]. Despite concerns over unregulated formulations and inconsistent results, oral glutathione may be used in small doses for patients with extensive pigmentation that resists treatment.

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Azelaic acid is a gentle skin treatment that reduces pigmentation through tyrosinase inhibition and inflammation reduction. The treatment of PIH and mild acanthosis nigricans (AN) in diabetes patients is effective, especially for those with skin of color. While it does not function as a direct AGE inhibitor, this substance helps alleviate glycation-related effects through its ability to decrease oxidative stress and lower IGF-1 (Insulin-like Growth Factor) activity. Due to its safety profile and dual mechanism, it serves as a practical treatment for hyperpigmentation on the neck and cheeks [32].

When prescribing topical therapies in SOC, it is essential to prevent skin irritation to avoid worsening PIH. A gradual introduction of treatments like retinoids, which are applied twice weekly and combined with hydrating vehicles, helps to minimize irritation risk. The combination treatment known as the Kligman formula remains the most effective standard method for addressing melasma, but can be carefully adjusted for use in diabetic pigmentation cases. Alternative treatments, such as azelaic acid, that target insulin-like growth factor pathways should be considered for PIH in SOC patients to prevent exogenous ochronosis from prolonged hydroquinone application.

Laser and Energy-Based Therapies

Treating hyperpigmentation through laser therapy in skin of color requires careful management because of the potential burn risks and PIH development. Contemporary advancements in medical technology now allow for relatively safe pigment treatment in higher phototypes. Q-switched Nd: Q-switched Nd: YAG lasers operating at a 1064 nm wavelength alongside new picosecond laser technology can target dermal melanin and hemosiderin while also breaking some advanced glycation cross-links based on experimental findings that specific laser wavelengths can cleave AGEs in collagen. Garg et al. (2024) reported successful treatment of 122 Indian patients with dermal hyperpigmentation through sequential laser sessions, which led to substantial pigment reduction with few side effects [30]. Using correct laser settings, such as reduced fluence and extended wavelengths with sufficient cooling, enables darker skin types to safely undergo laser toning for the reduction of diabetic-related pigmentation, including persistent PIH or AN.

The 1550 nm erbium fiber and 1927 nm thulium nonablative fractional lasers generate controlled thermal injury columns that drive pigment removal and collagen restructuring, potentially reducing dermal glycation stiffness [30]. The successful application of these lasers for scarring and hyperpigmentation treatment in colored skin suggests potential effectiveness for AN plaque when metabolic causes are resolved [17]. Long-pulse lasers and Intense Pulsed Light (IPL) devices effectively manage erythema and superficial pigment. Yet, IPL treatment poses significant risks for Fitzpatrick types V–VI because of increased melanin absorption and elevated adverse effect incidence [10]. With growing experience and refined protocols for SOC, these technologies emerge as promising tools for managing stubborn pigmentation and textural changes in diabetic skin (Figure 2).

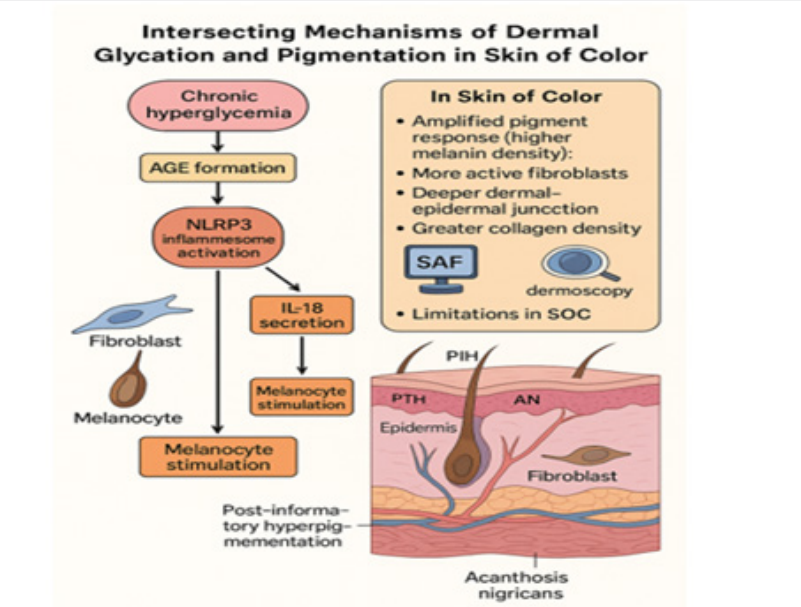

Figure 2: Intersecting Mechanisms of Dermal Glycation and Pigmentation in Skin of Color.
This conceptual diagram compares glycation and pigmentation pathways in skin of color (SOC), highlighting intensified responses due to greater fibroblast activity, collagen density, and melanin levels. It also illustrates limitations of current diagnostic devices like the SAF reader in darker phototypes and includes emerging targets for pigment modulation [3,5,13].

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Systemic Therapies

Diabetic patients can gain skin-related health benefits through systemic treatments that exceed the glycation-reducing effects of glucose-lowering medications like metformin. The lipid-soluble vitamin B1 derivative benfotiamine can block various glycation pathways, lowering tissue AGE buildup and inflammatory responses. Research has not yet been conducted to determine skin effects, but benfotiamine could enhance skin suppleness and wound healing by reducing oxidative stress [24]. ACE inhibitors and ARBs used for hypertension treatment suppress AGE formation by reducing oxidative stress and enhancing microcirculatory function. Research shows their use leads to better collagen restructuring and wound repair in diabetic ulcers, which implies secondary advantages for skin health [25]. People with skin of color and diabetes experience high rates of vitamin D deficiency. The presence of vitamin D enhances the skin's immune system while potentially influencing insulin resistance levels. Though direct pigmentary outcome research is scarce, appropriate vitamin D levels can decrease inflammation and skin infections, which lead to post-inflammatory hyperpigmentation [32].

Innovative "Glycation Reversers"

Research is advancing innovative strategies for reversing glycation by developing compounds that break existing preformed AGE crosslinks. Clinical trials demonstrated that the agent alagebrium (ALT-711) could potentially decrease arterial stiffness. Breaking AGE-induced collagen crosslinks has the potential to restore skin elasticity and dermal function, even though it currently lacks direct application in dermatology [36]. Topical delivery continues to be a principal obstacle, but upcoming treatments might strive to reverse glycation in skin structural proteins.

The work of Byun et al. (2024) provides an interesting supplementary finding. This study showed that injecting poly-L-lactic acid (PLLA) into photoaged skin maintained the dermal–epidermal junction and prevented UVB-induced pigmentation through basement membrane preservation. The research on photoaging reveals that the mechanism that strengthens dermal matrix architecture to block pigmentary alterations could also be helpful for diabetes treatment [33]. Applying bio stimulatory fillers or comparable techniques to boost dermal resilience might hypothetically prevent some skin changes associated with diabetes. Despite being speculative, research identified the connection between skin structure and pigment control during aging and glycation.

Preventive Skin Care Strategies in Diabetic Patients with Skin of Color

Skin care preventive measures remain essential yet overlooked in diabetes treatment, especially for skin of color patients who experience persistent and distressing post-inflammatory hyperpigmentation. Applying broad-spectrum sunscreen with SPF 30 or higher daily helps prevent UV-induced pigmentation changes and oxidative skin damage. SOC patients experience less skin cancer risk but need to protect against UV and visible light, which worsen pigmentation disorders like melasma and PIH. Iron oxide-based physical sunscreens provide exceptional protection by blocking visible light that triggers melanogenesis [10,17].

Emollients for skin hydration are crucial for maintaining optimal skin health. Xerosis affects diabetic patients who can benefit from the regular application of ceramide or urea moisturizers to minimize microtrauma and itching while lowering the chances of developing ulcers or PIH [25]. People with diabetes must practice proper foot care to avoid developing ulcers, which can risk becoming chronic wounds or causing scars and pigmentary complications. Protective footwear, regular check-ups, and immediate intervention for skin disorders such as tinea pedis or hyperkeratosis form part of the foot care routine [32].

Educating patients about steering clear of tough or unregulated over-the-counter products is crucial. The frequent misuse of skin lightening products that contain steroids or mercury can cause exogenous ochronosis and worsen post-inflammatory hyperpigmentation. Medical professionals should direct patients to proven gentle treatments, while dermatologists oversee these procedures [30].

Personalized medicine requires adaptation of treatments to align with individual skin type characteristics and cultural practices. An African American patient suffering from AN could receive insulin resistance benefits from metformin, together with pigment fading from a well-tolerated retinoid. In contrast, an Asian patient with a propensity for PIH will likely see better results from niacinamide or thiamidol, which causes less irritation. When patients participate in shared decision-making and their psychosocial concerns about skin changes receive recognition, they exhibit better treatment adherence and outcomes. While some people regard AN as merely a cosmetic concern, others understand it as an indicator of metabolic dysfunction. Patients who experience psychological distress from darkened skin can find comfort and encouragement through setting attainable treatment schedules that demonstrate visible results over several months [23].

A holistic, interdisciplinary approach is ideal. Dermatologists are responsible for detecting skin indicators of metabolic disorders like new-onset AN and making proper referrals for diabetes screening. When treating patients with diabetes, endocrinologists must perform regular skin examinations and refer patients with unusual skin lesions or those resistant to treatment to dermatologists. The collaborative care model proves especially beneficial for SOC patients because they encounter distinct diagnostic and therapeutic obstacles while facing healthcare disparity risks [15,25].

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Future treatment approaches should focus on developing natural antiglycation substances and botanical extracts that offer potential benefits in pigment regulation. Research shows that Terminalia arjuna bark extract protects proteins from glycation and simultaneously reduces melanogenesis in experimental settings [13]. These agents serve as cost-efficient supplemental choices for areas with limited resources or individuals who choose plant-based treatments. The development of skin-focused antiglycation therapies could lead to the normalization of removing "sugar damage" from skin alongside the common practice of retinoid use for photoaging treatment.

In summary, managing dermal glycation and hyperpigmentation in diabetes is a two-pronged strategy: Treat patients by managing their metabolic state through glucose regulation and diet alteration while concurrently using secure topical treatments and procedural methods to directly treat skin conditions with a focus on patient-centered preventive care. Healthcare providers prioritize clinical treatment and cultural sensitivity when treating patients with skin of color by reducing unwanted pigmentation and improving overall skin appearance. Through regular treatment, many skin conditions from diabetes show improvement, often as visible indicators of internal health progress, like AN fading due to better insulin sensitivity.

Discussion

This review combines existing research on dermal glycation and hyperpigmentation in patients with skin of color and type 2 diabetes mellitus while highlighting both advancements and remaining gaps in clinical knowledge. The study shows that although diabetes can present skin symptoms like acanthosis nigricans, diabetic dermopathy, and scleroderma across different ethnic groups, these skin manifestations appear differently and require distinct diagnostic criteria for darker skin tones. The terms "erythematous base" and "pink lesion" do not accurately describe presentations in SOC because these signs often appear as violaceous, brown, or invisible to the naked eye [9]. Underdiagnosis continues to be a significant issue in primary care settings, according to research from both Walha et al. (2024) and Svoboda & Shields (2021) [7,9]. The causes are multifactorial: Medical professionals lack dermatologic training for different skin types, while patients fail to report symptom-free skin changes. Healthcare providers lack the time to prioritize skin examinations. To address this healthcare gap, medical education should include diverse imagery and teach students to recognize skin changes as early indicators of metabolic issues. Education at the community level helps patients to identify early diabetic symptoms such as darkening of the neck and foot dryness (Figure 3).

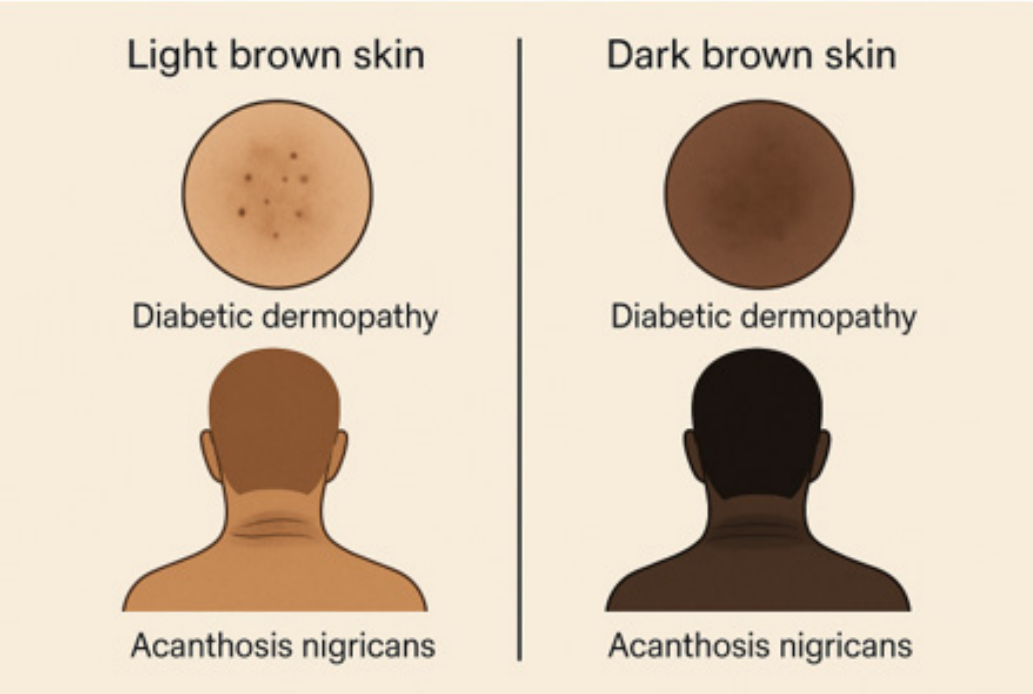

Figure 3: Clinical Presentation of Diabetic Dermatoses in Light Brown vs. Dark Brown Skin.
This clinical image comparison displays common diabetic skin findings such as acanthosis nigricans, diabetic dermopathy, and post-inflammatory hyperpigmentation, highlighting how these presentations differ in visibility between lighter and darker skin tones. The figure underscores the diagnostic challenge and the need for heightened clinical vigilance in SOC [7-9,25].

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This literature review highlights the significant role of advanced glycation end products in altering skin properties, leading to pigmentary imbalances. AGEs are not only metabolic byproducts, but they also act as active agents of inflammation and contribute to oxidative stress and the degradation of the dermal matrix. Research conducted by Lee et al. (2016) indicates that these processes can disrupt melanogenesis and collagen remodeling [1]. Further studies by Fang et al. (2024) and Lan et al. (2025) demonstrate that targeting the formation of AGEs and their receptor binding through specific interventions can reduce systemic complications while improving skin tone and texture [3,5]. For instance, Zhang et al. (2025) found that higher levels of AGEs in the skin are linked to increased skin yellowness, suggesting that visible skin pigmentation could be an indicator of glycemic history [2]. Healthcare providers can leverage visible improvements in skin appearance to encourage patients to pursue better glycemic control.

This review highlights the limitations of existing diagnostic tools and scoring systems. SAF readers have proven effective in lighter-skinned populations, but their performance decreases with skin of color due to the absorption of fluorescence signals by melanin. A study by Ahdi et al. (2015) found that poor reflectance values excluded one-third of Black African subjects from SAF analyses. This underscores a broader issue: diagnostic devices, dermatologic scoring systems (such as PASI (Psoriasis Area and Severity Index) and SCORAD (Scoring Atopic Dermatitis)), and risk stratification algorithms need to be redeveloped for appropriate use across diverse ethnicities [19]. Recent validation efforts of advanced tools show promise, as they are being tested in various population groups. For example, the smartphone-based AN grading system researched by Dhanoo et al. (2024) is one such tool [29]. Additionally, Pagan et al. (2022) reviewed diagnostic methods that integrate dermoscopy and confocal microscopy, specifically adapted for pigmentary conditions in SOC [10].

Clinical issues arise as part of the broader healthcare disparity framework. African American, Hispanic, and South Asian populations experience higher T2DM rates because of genetic risks combined with social factors, which limit their access to nutritious food, healthcare, and educational opportunities [15,25]. These communities experience restricted access to specialty medical services such as dermatological care. Patients with SOC receive regular check-ups from endocrinologists but typically lack referrals to dermatologists even when skin symptoms such as acanthosis nigricans and pigmented skin lesions occur. The lack of treatment or mismanagement through over-the-counter remedies leaves these conditions untreated. Abate et al. (2025) demonstrate that dermatologic complications of diabetes often go unnoticed, even though they hold significant prognostic importance [23]. Under-served SOC populations could benefit from early skin sign detection through interdisciplinary models that combine dermatologic screening with diabetes management clinics or community-based outreach programs such as barbershops and churches.

Research to evaluate the effects of treating diabetic skin conditions on psychosocial or metabolic outcomes has yet to be conducted, but remains an important field for investigation. Patients who notice physical improvements in AN reportedly gain confidence, which might lead to a greater inclination to adopt healthy lifestyle changes, including exercise. The cultural significance of skin appearance suggests that treating visible pigmentation might contribute to metabolic benefits, which requires additional research [32].

Table 3: Evidence Gaps and Research Priorities in Dermal Glycation and Hyperpigmentation in Skin of Color.

Gap	Description	Research Direction
Lack of device validation [10,19]	Diagnostic tools like SAF and RCM perform sub optimally in SOC due to melanin interference	Validate tools in Fitzpatrick IV-VI cohorts; explore melanin-neutral alternatives
Limited mechanistic studies in ethnic skin [5,6]	Most in vitro models use light-skin-derived cell lines	Use fibroblasts/melanocytes from diverse populations; examine genetic polymorphisms
Underrepresentation in clinical trials [17,30]	Few trials assess pigmentation therapies specifically in SOC diabetic patients	Stratify trials by skin type; test anti-AGE agents and pigment lighteners in SOC
Minimal psychosocial and stigma research [29,32]	Cultural stigma around pigmentation changes underexplored	Conduct qualitative studies on impact, perception, and healthcare-seeking behaviors
Extrapolation from non-diabetic pigmentation studies [15,32]	Many therapies are borrowed from general hyperpigmentation treatments	Design SOC-specific diabetic pigmentation protocols (e.g., for AN, dermopathy)
Few longitudinal studies [15,25]	No large-scale cohort data on progression of diabetic skin changes in SOC	Establish cohorts to map onset, severity, and correlations with glycemic control

Abbreviations: SAF- Skin Autofluorescence; RCM- Reflectance Confocal Microscopy; SOC- Skin of Color; AGE- Advanced Glycation End Products; AN- Acanthosis Nigricans; PIH- Post-Inflammatory Hyperpigmentation.

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The review exposes a deficiency in high-quality evidence that guides treatment approaches in SOC. Current research indicates that while thiamidol and low-fluence lasers, along with antioxidant-based cosmeceuticals, show promise, research trials often include limited participant numbers and rarely focus on diabetic populations or skin diversity [17,30]. Subsequent studies need to categorize participants by skin type and ethnicity while assessing both cosmetic and metabolic results. A placebo-controlled study using topical anti-AGE agents in African-American patients suffering from AN will produce actionable insights and specific treatment guidelines (Table 3).

Dermatologic treatment plans for diabetic patients should incorporate genetic pigmentation differences as well as cultural practices, environmental factors, and metabolic health profiles. Patients suffering from metabolic syndrome and SOC should consider a combined treatment plan that includes GLP-1 (glucagon-like peptide) agonists for metabolic control alongside metformin which provides both metabolic and skin benefits and topical triple-combination creams to address localized pigmentation issues [32]. Collaboration between endocrinologists and dermatologists is increasingly emphasized, as shown in emerging multidisciplinary frameworks and publications like Springer's Cutaneous Manifestations in Diabetes textbook, which includes contributions by Fritz et al. (2024) and Staubach & Staubach (2024) [15,24].

The study of how dermal glycation affects skin pigmentation in patients with SOC and diabetes stands as an important and fruitful clinical research domain. Diagnostic approaches need modification to account for differences in skin color and systemic treatments must evaluate their effects on skin health while inclusive investigations must be prioritized to address healthcare disparities. Knowledge and culturally sensitive care tools for clinicians and patients enhance skin and systemic outcomes in high-risk groups.

Limitations

While this review aimed to synthesize the intersection of dermal glycation, hyperpigmentation, and type 2 diabetes in skin of color, several limitations must be acknowledged. First, the current body of literature remains limited in scope and depth when it comes to skin type-specific data. Many referenced studies do not stratify findings by Fitzpatrick skin type, ethnicity, or pigmentation phenotype, which restricts the ability to draw definitive conclusions for SOC populations. For example, pigmentation-related outcomes are often described in general terms without specifying whether participants had darker phototypes, leaving gaps in interpretation [15,32].

Second, a number of mechanistic insights regarding glycation and pigmentation come from in vitro models or murine studies, which may not fully replicate human cutaneous physiology-especially in ethnic skin. While some studies include skin biopsy or autofluorescence data, few directly compare cellular or structural glycation differences between ethnic groups. Similarly, patient-reported outcomes, cultural influences on treatment preferences, and psychosocial impacts remain largely unexplored in formal clinical trials.

Third, some of the insights presented in this review involve extrapolation-for instance, using data from photoaging, metabolic syndrome, or general pigmentation studies and applying them to diabetic skin of color. While this approach is necessary given the paucity of focused literature, it introduces potential interpretative bias. Additionally, while this review includes high-quality sources from 2015-2025, the overall number of randomized controlled trials or longitudinal cohort studies in this domain remains low, which limits the strength of evidence guiding practice in SOC patients with diabetes-related skin disorders.

Lastly, device-based tools such as skin autofluorescence readers, dermoscopy, and reflectance confocal microscopy have been evaluated primarily in light-skinned populations. Their reliability and diagnostic thresholds may not translate to SOC, and there is insufficient published validation for these tools in darker skin types [10,19]. As a result, clinical decisions made using these technologies may be skewed or less accurate in ethnically diverse populations.

Future Research Directions

To advance equitable dermatologic care in diabetes, future research must address these limitations directly. A primary area of need is the inclusive validation of diagnostic technologies. Devices such as the AGE Reader™ must be re-evaluated for accuracy in patients with higher melanin content. Alternatives such as Raman spectroscopy, which is less sensitive to skin pigmentation, may provide more reliable AGE measurement in SOC [19,27].

Second, mechanistic studies should explore ethnicity-specific differences in skin response to hyperglycemia and glycation. For example, following the findings of Lan et al. (2025) on the YTHDF2/A20/IL-18 axis in melanogenesis, future research could investigate whether fibroblasts or melanocytes from African-origin individuals express more IL-18 or are more responsive to AGE stimuli compared to those from lighter-skinned individuals [5]. Genomic studies exploring polymorphisms in glycation and inflammation pathways across ethnic groups may help explain differing pigmentary and structural outcomes.

Third, longitudinal cohort studies involving SOC patients with T2DM are needed to track the natural history of cutaneous changes, correlate them with metabolic markers, and refine screening guidelines. For instance, if AN consistently presents during prediabetes in Black or Hispanic patients, it may warrant inclusion in diabetes risk assessment algorithms [15,32].

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Interventional research is necessary to explore various treatment options. Clinical trials should focus on both pharmacological and procedural therapies, including antioxidant supplements like rosemary extract, topical agents that combat advanced glycation end products, and pigment-targeted treatments such as thiamidol, low-fluence lasers, or carnosine creams. The studies conducted by Draelos et al. (2025) and Guiotto et al. (2025) show promising improvements in oxidative stress markers and skin dullness with rosemary extract. However, larger trials involving standard of care populations are crucial for further validation [11,12].

Additionally, psychosocial research should not be overlooked. Qualitative studies examining how skin changes impact the self-perception, stigma, and healthcare engagement of SOC patients with diabetes could uncover new avenues for culturally tailored education and support. For instance, stigma surrounding neck darkening in AN may lead to social withdrawal or inappropriate use of bleaching creams. Understanding these dynamics is key to improving adherence and quality of life.

This review highlights that patients with skin of color and type 2 diabetes represent a population where cutaneous signs are both clinically relevant and often underappreciated. Recognizing these dermatologic manifestations-such as post-inflammatory hyperpigmentation, acanthosis nigricans, and dermopathy-not only enhances patient comfort but also serves as a potential window into systemic metabolic health. The dual processes of advanced glycation and melanogenesis, influenced by inflammatory pathways and pigment cell responses, offer unique targets for both systemic and topical intervention.

As precision medicine and culturally competent care continue to evolve, clinicians must integrate skin tone–specific considerations into their diagnostic and therapeutic approaches. No patient's symptoms should be dismissed or misinterpreted due to the color of their skin. Integrating dermatologic surveillance into diabetes care, validating tools for diverse populations, and tailoring therapies to ethnic and cultural context are all steps toward reducing disparities and optimizing outcomes.

Ultimately, bridging dermatology and endocrinology holds the potential not only to treat diabetic skin changes but also to empower earlier diagnosis and improved metabolic control. Continued multidisciplinary collaboration and inclusive research will ensure that skin of color is not an afterthought in diabetes care, but a focus-where what's visible on the surface can lead to deeper healing within.

Conclusion

Type 2 diabetes patients with skin of color display specific skin alterations that result from chronic hyperglycemia and glycation processes. The presence of high levels of melanin and specific skin structure affects the impact of advanced glycation end products by causing noticeable hyperpigmentation, such as AN and post-inflammatory darkening, along with skin stiffening while reducing the visibility of traditional signs like erythema or wrinkling. The need for individualized clinical awareness and diagnostic strategies becomes critical in SOC populations because diabetic rashes that stand out on lighter skin are often masked in darker skin tones. There is ongoing concern about missing diabetic skin manifestations in SOC, which may postpone necessary medical responses.

Identifying signs of diabetes on skin regardless of tone allows for prompt metabolic testing and treatment, which serves as a "clinical clue" for hidden insulin resistance or elevated blood sugar levels. Patient care improves significantly when glycemic control helps reduce dermal glycation alongside skin-directed therapies for hyperpigmentation and new anti-glycation treatments. The effective treatment of disfiguring pigmentary lesions on skin of color with topical tyrosinase inhibitors and antioxidants alongside laser therapy requires understanding the skin's sensitivities. Integrating preventive skincare alongside patient education into diabetes management programs enables patients to better care for their skin, which may help prevent complications like PIH from minor injuries.

This review showcases how dermatology intersects with endocrinology and points out healthcare providers' need to deliver multidisciplinary care that respects cultural differences. Healthcare providers can enhance patient outcomes and diminish disparities by recognizing ethnic variations in skin presentation when calibrating diagnostic and therapeutic tools. The skin changes associated with diabetes represent actionable signs rather than unavoidable cosmetic issues. Addressing skin changes in diabetes patients can enhance their living standards and may lead to better compliance with their comprehensive diabetes care regimen.

The skin changes caused by dermal glycation and hyperpigmentation demonstrate an observable connection between biochemical processes and physical symptoms in diabetes. Understanding this connection in patients with skin color variations enables earlier diagnosis and treatment customization, along with comprehensive patient care. Research should prioritize diverse population inclusion to ensure that all people benefit from advances in diagnosing and treating glycation-related skin damage. This approach allows us to respect both scientific and humanistic medical principles by understanding patients as complete individuals who require comprehensive care and sensitive handling of their complex health needs.

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