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Diabetic Fasciopathy Linking Chronic Hyperglycemia and Advanced Glycation End Products to Musculoskeletal Dysfunction

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Abstract
Diabetes-related injury to fascia had received less attention than vascular and neurological complications despite its potential contribution to musculoskeletal dysfunction. This narrative review examined the relationship between chronic hyperglycemia, advanced glycation end products (AGEs), fascial remodeling, and clinical impairment. A targeted literature search emphasized human imaging and tissue studies, mechanistic reviews, and clinical guidelines. Evidence was synthesized thematically, with established associations distinguished from proposed mechanisms. Human studies consistently reported increased plantar fascia thickness and associations with glycemic exposure, neuropathy, microvascular disease, and altered plantar loading. Direct tissue analysis demonstrated site-specific nanoscale stiffening, whereas some ultrasound elastography studies reported lower macroscopic stiffness. These differences suggested that anatomical location, measurement scale, and tissue organization influenced mechanical findings. AGE-related collagen modification, oxidative stress, inflammation, and extracellular matrix remodeling provided a plausible biological framework, but direct evidence for diabetes-specific impairment of hyaluronan-mediated gliding remained limited. No validated diagnostic criteria or fascia-specific treatment pathway were identified in the reviewed literature. The review supported diabetic fasciopathy as an emerging explanatory framework rather than an established independent diagnosis. Clinical implications favored comprehensive metabolic care, preservation of mobility, management of regional disorders, and guideline-directed diabetic foot protection. Prospective studies were needed to establish diagnostic thresholds, incremental prognostic value, and treatment responsiveness.
Keywords: *tissue glycation; ultrasonography; elastography; joint mobility; diabetic foot; connective tissue*

INTRODUCTION

Diabetes mellitus causes multisystem injury, but conventional descriptions of its complications emphasize vascular disease, neuropathy, nephropathy, retinopathy, and impaired wound healing. Connective-tissue manifestations receive less attention despite their potential effects on mobility, pain, and independence.

Fascia is a layered, collagen-rich extracellular matrix network involved in force transmission, tissue sliding, and sensory signaling (Kwong & Findley, 2014; Stecco et al., 2025). Hyaluronan-rich interfaces facilitate movement between adjacent layers (Pratt, 2021; Stecco et al., 2011). Long-lived matrix proteins are susceptible to non-enzymatic glycation; chronic hyperglycemia can therefore alter collagen chemistry, inflammatory signaling, and tissue mechanics (Brownlee, 2001; Brownlee, 2005; Khalid et al., 2022; Suzuki et al., 2022).

Diabetic fasciopathy is used here as a conceptual term for diabetes-associated structural, biochemical, and biomechanical abnormalities of fascia. It is not a standardized diagnosis. Its proposed value is to integrate findings otherwise discussed separately, including plantar fascia thickening, altered loading, and connective-tissue stiffness. Recent human tissue research provides direct evidence of regional fascial remodeling (Ugwoke et al., 2026), while broader mechanometabolic models remain partly theoretical (Ferguson, 2026).

This review aimed to connect biological mechanisms with human evidence and clinical implications, while distinguishing established observations from hypotheses. Its contribution was an integrated interpretation of imaging, tissue mechanics, and functional manifestations that avoided treating all diabetic connective-tissue disorders as a single proven disease.

METHOD

A targeted narrative literature review emphasized PubMed-indexed human studies, mechanistic reviews, and major clinical guidelines. Search concepts included diabetes

mellitus, fascia, plantar fascia, advanced glycation end products, extracellular matrix, elastography, diabetic foot, limited joint mobility, adhesive capsulitis, and tendinopathy. Recent human fascial investigations were prioritized, with established literature retained for biological and clinical context.

The evidence was organized into mechanisms, human fascial findings, clinical manifestations, assessment, and management. Interpretation distinguished observational associations, direct tissue measurements, and extrapolation from related connective tissues. Clear aims, transparent description of the review approach, and scientific reasoning are recognized quality domains for narrative reviews (Baethge et al., 2019). This review was not a systematic review or meta-analysis. Exact search dates, full search strings, screening counts, and formal risk-of-bias assessments were not available; completeness and reproducibility were consequently limited.

RESULTS AND DISCUSSION

Biological basis and regional tissue remodeling

Non-enzymatic glycation produces advanced glycation end products (AGEs), which can modify collagen through cross-linking and reduced turnover. AGE interaction with the receptor for advanced glycation end products (RAGE) also promotes oxidative stress and inflammatory signaling (Brownlee, 2001; Brownlee, 2005; Khalid et al., 2022; Suzuki et al., 2022). These mechanisms offer a plausible explanation for reduced adaptability of mechanically active connective tissue, although their precise contribution to human fascial dysfunction remains incompletely quantified.

Fascial extracellular matrix may undergo fibrosis, abnormal deposition, altered hydration, and fibroblast dysfunction. Tendon studies provide parallel evidence of glycation-related collagen disorganization and impaired healing (Lui, 2017; Shi et al., 2021; Xu et al., 2024), but tendon findings cannot be assumed to establish identical fascial pathology. Similarly, hyaluronan normally supports fascial sliding (Pratt, 2021; Stecco et al., 2011), yet diabetes-specific hyaluronan aggregation and clinically important impairment of gliding remain insufficiently demonstrated.

Ugwoke and colleagues examined deep fascia from 17 male donors with type 2 diabetes and 17 controls using atomic force microscopy and multiphoton imaging (Ugwoke et al., 2026). Plantar fascia and fascia lata showed increased nanoscale stiffness, whereas thoracolumbar fascia did not show a significant difference. Imaging also revealed biochemical changes compatible with collagen modification. These findings support regional susceptibility rather than uniform stiffening throughout the fascial system. Figure 1 summarizes the proposed pathway and its uncertainties.

Human imaging and biomechanical evidence

Plantar fascia thickness (PFT) provides the most consistent human evidence. D'Ambrogi et al. associated increased thickness with reduced first metatarsophalangeal mobility and greater forefoot forces (D'Ambrogi et al., 2003). Giacomozzi et al. reported plantar fascia and Achilles tendon thickening alongside altered diabetic foot loading (Giacomozzi et al., 2005). Together, these studies suggest that connective-tissue changes may modify the mechanical environment in which neuropathy and repetitive pressure act.

In 344 adolescents with type 1 diabetes, Craig et al. found that PFT predicted subsequent microvascular complications (Craig et al., 2008). Longitudinal findings from Benitez-Aguirre et al. linked PFT with retinopathy and renal dysfunction (Benitez-Aguirre et al., 2012). These associations support investigation of PFT as a marker of cumulative metabolic exposure, but do not establish additional clinical value beyond conventional risk assessment.

Ultrasound and elastographic investigations repeatedly demonstrated increased PFT (Bozkus et al., 2025; Harish et al., 2022; Saroha et al., 2023; Wang et al., 2025). Saroha et al., Wang et al., and Bozkus et al. also reported lower shear-wave measurements or stiffness in diabetic groups (Bozkus et al., 2025; Saroha et al., 2023; Wang et al., 2025). Such results appear different from nanoscale stiffening in cadaveric tissue (Ugwoke et al., 2026). Atomic force microscopy assesses local material behavior ex vivo, whereas elastography measures wave propagation through composite living tissue. Fiber orientation, loading, hydration, thickness, and measurement protocols can influence their results. Coexisting microscopic stiffening and macroscopic disorganization remain a possible explanation, not a directly established reconciliation.

Casadei et al. studied 290 adults with type 1 diabetes and 41 controls (Casadei et al., 2026). Higher PFT was associated with worse glycemic control, longer disease duration, greater skin autofluorescence-derived AGE burden, reduced vibration sensitivity, retinopathy, impaired renal function, and worse foot-risk classification. Cross-sectional associations cannot establish causality or treatment effects. In chronic Charcot diabetic foot, Pirri et al. observed plantar and crural fascial abnormalities (Pirri et al., 2023), which indicate fascial involvement without showing that fascia initiates neuro-osteoarthropathy. Table 1 summarizes selected human studies.

Clinical manifestations and diabetic foot relevance

Plantar fasciitis has been reported more frequently among people with diabetes, particularly type 2 diabetes (Gariani et al., 2020; Priesand et al., 2019). Obesity, altered loading, neuropathy, and restricted mobility may contribute alongside metabolic matrix injury. Asymptomatic fascial thickening should not automatically be classified as symptomatic plantar fasciopathy.

Limited joint mobility and diabetic cheiroarthropathy illustrate clinically important stiffness of collagen-rich periarticular tissues (Gerrits et al., 2015). Diabetes is also strongly associated with adhesive capsulitis (Zreik et al., 2016). These conditions involve structures beyond fascia and should retain their established diagnostic identities. Tendinopathy similarly belongs to a related connective-tissue spectrum rather than being relabeled as fasciopathy (Lui, 2017; Shi et al., 2021; Xu et al., 2024).

Fascial innervation provides an anatomical basis for participation in pain (Gromakovskis, 2025), but diabetic musculoskeletal symptoms remain multifactorial. Burning, electric pain and distal sensory disturbance suggest neuropathic mechanisms, whereas focal tenderness and movement-related restriction suggest regional musculoskeletal disease. Both may coexist, requiring examination rather than attribution to fascia alone.

In the foot, reduced joint mobility and altered plantar fascia mechanics may increase repetitive pressure (D'Ambrogi et al., 2003; Giacomozzi et al., 2005). With sensory loss, such stress could contribute to tissue injury. However, fascial abnormalities have not been established as independent causal predictors of ulceration. Assessment should continue to prioritize protective sensation, peripheral arterial disease, deformity, callus, and previous ulceration or amputation (American Diabetes Association Professional Practice Committee for Diabetes, 2026; Schaper et al., 2024). Table 2 outlines the proposed clinical framework and its evidence limits.

Assessment and management implications

No validated diagnostic criteria or universal imaging thresholds exist for diabetic fasciopathy. Clinical assessment should address diabetes duration, glycemic control, stiffness, regional pain, walking capacity, prior ulcers, neuropathy, and vascular disease. Examination can assess joint motion, prayer or tabletop signs, plantar tenderness, ankle dorsiflexion, gait, footwear, and pressure-related skin changes. Standard diabetic foot examination remains

essential (American Diabetes Association Professional Practice Committee for Diabetes, 2026; Gerrits et al., 2015; Schaper et al., 2024).

Ultrasound can characterize fascial thickness and architecture; elastography may add mechanical information (Bozkus et al., 2025; Harish et al., 2022; Saroha et al., 2023; Wang et al., 2025). Neither should replace established clinical assessment. Device differences, anisotropy, joint position, loading, and operator technique limit interpretation of isolated stiffness measurements. Standardization and prospective validation are required before imaging can define a distinct diagnosis.

Management should address recognized diabetes-related risks and specific symptomatic disorders. Associations between glycemic exposure and PFT reinforce metabolic care (Benitez-Aguirre et al., 2012; Casadei et al., 2026; Craig et al., 2008), but reversal of established collagen cross-links has not been demonstrated. Mobility exercises, appropriate activity, and rehabilitation can address functional restrictions; plantar fasciopathy and other regional disorders require diagnosis-specific care (Gariani et al., 2020; Gerrits et al., 2015).

Patients at risk of diabetic foot disease require surveillance, suitable footwear, pressure management, and offloading when indicated (American Diabetes Association Professional Practice Committee for Diabetes, 2026; Schaper et al., 2024). Manual or myofascial techniques may be considered within rehabilitation, but claims that they remove AGEs or reverse pathological collagen cross-linking are unsupported. AGE-pathway modulation remains investigational for fascial disease (Khalid et al., 2022; Suzuki et al., 2022). No fascia-specific pharmacological treatment was established in the reviewed evidence.

Evidence limitations and research priorities

This synthesis combined heterogeneous observational studies, cadaveric measurements, related connective-tissue research, and conceptual models. Findings differ by anatomical region, diabetes type, clinical severity, and measurement scale. Selection bias and residual confounding by obesity, age, neuropathy, activity, or vascular disease may affect associations. The non-systematic search also limits certainty that all relevant evidence was captured.

Future studies should establish reproducible phenotypes, standardized imaging protocols, and clinically meaningful thresholds. Multiscale research should relate biochemical glycation to histology, tissue mechanics, elastography, and function. Longitudinal studies must test whether fascial measurements improve prediction beyond established markers, while intervention trials should evaluate responsiveness to metabolic care and rehabilitation. Proposed systemic mechanometabolic functions of fascia require further validation (Ferguson, 2026).

Table 1. Selected human evidence for diabetic fascial remodeling

Study	Population / method	Main finding	Interpretation
D'Ambrogi et al. (2003)	Diabetes vs controls; ultrasound and plantar biomechanics	Increased PFT; association with reduced MTP mobility and increased forefoot force	Fascial remodeling may influence diabetic foot mechanics
Giacomozzi et al. (2005)	Diabetes with/without neuropathy vs controls	Plantar fascia and Achilles tendon thickening	Connective-tissue changes contribute to rigid foot mechanics

Study	Population / method	Main finding	Interpretation
Craig et al. (2008)	344 adolescents with T1DM	PFT predicted later microvascular complications	Possible marker of cumulative tissue glycation
Benitez-Aguirre et al. (2012)	Longitudinal T1DM cohort	PFT associated with retinopathy and renal dysfunction	Supports systemic metabolic association
Harish et al. (2022)	T2DM vs controls; ultrasound/elastography	Increased PFT and altered elasticity	Non-invasive detection feasible
Saroha et al. (2023)	T2DM vs controls; SWE	Thicker PF with lower shear-wave velocity	Macro-scale viscoelastic abnormality
Pirri et al. (2023)	Chronic Charcot foot vs controls	Crural and plantar fascia abnormalities	Fascial involvement in advanced diabetic foot phenotype
Wang et al. (2025)	T2DM with heel pain; US + SWE	Increased PFT, reduced shear-wave velocity	Imaging markers distinguish diabetic PF abnormalities
Bozkus et al. (2025)	172 T2DM + 117 controls	Greater thickness and lower stiffness, worsening with neuropathy/ulcer	Association with diabetes severity and foot complications
Ugwoke et al. (2026)	Human cadaveric fascia; AFM + multiphoton imaging	Site-specific nanoscale stiffening and biochemical remodeling	Direct tissue-level evidence
Casadei et al. (2026)	290 T1DM + 41 controls	PFT associated with HbA1c, AGE burden, neuropathy and microvascular disease	Potential metabolic phenotyping role

Abbreviations: AFM, atomic force microscopy; AGE, advanced glycation end product; MTP, metatarsophalangeal; PF, plantar fascia; PFT, plantar fascia thickness; SWE, shear-wave elastography; T1DM, type 1 diabetes mellitus; T2DM, type 2 diabetes mellitus; US, ultrasound.

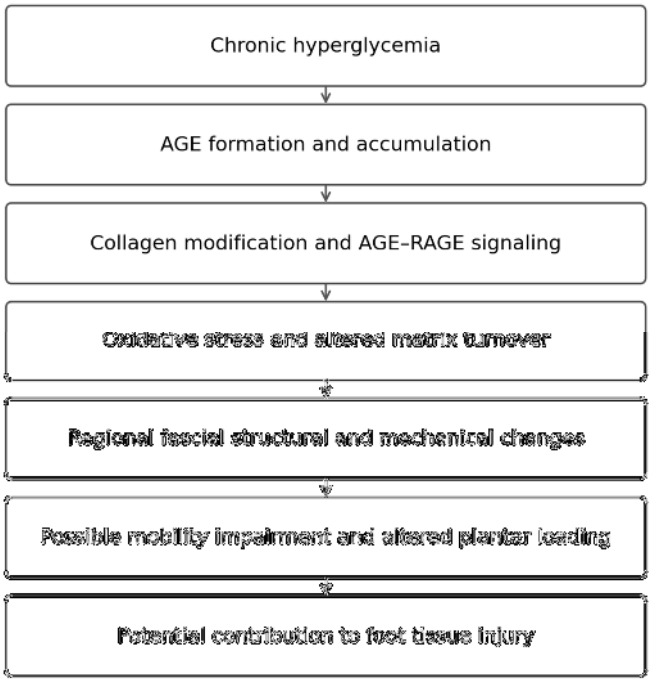
Table 2. Proposed clinical framework and evidence limits

Domain	Potential abnormality	Possible clinical consequence	Current clinical status
Collagen	AGE cross-linking and altered turnover	Tissue stiffness, impaired adaptation	Strong biological rationale
Extracellular matrix	Fibrotic remodeling	Reduced tissue compliance	Supported by related connective-tissue evidence

Domain	Potential abnormality	Possible clinical consequence	Current clinical status
Plantar fascia	Increased thickness	Reduced MTP mobility, altered loading	Repeated human evidence
Fascial mechanics	Region-specific stiffness/viscoelastic changes	Abnormal force transmission	Emerging evidence
Periarticular tissue	Increased stiffness/fibrosis	Cheiroarthropathy, limited ROM	Established diabetic association
Shoulder capsule	Fibrosis and contracture	Adhesive capsulitis	Strong epidemiological association
Foot biomechanics	Increased plantar pressure	Potential contribution to ulcer risk	Mechanistically plausible; not an independent validated risk marker
Nociceptive system	Mechanical/inflammatory input	Regional musculoskeletal pain	Emerging; multifactorial
Ultrasound PFT	Increased thickness	Possible cumulative glycation marker	Promising but not validated for routine screening

Abbreviations: AGE, advanced glycation end product; MTP, metatarsophalangeal; PFT, plantar fascia thickness; ROM, range of motion.

Figure 1. Proposed pathway of diabetic fascial remodeling



The pathway is a conceptual synthesis, not a validated causal sequence. Dashed arrows indicate proposed downstream relationships. Effects may vary with anatomical site, loading, diabetes duration, glycemic exposure, age, obesity, neuropathy, and vascular disease. Impaired hyaluronan-mediated gliding remains hypothetical. AGE, advanced glycation end product; RAGE, receptor for advanced glycation end products. Based on (Brownlee, 2001; Brownlee, 2005; D'Ambrogi et al., 2003; Giacomozzi et al., 2005; Khalid et al., 2022; Pratt, 2021; Stecco et al., 2011; Suzuki et al., 2022; Ugwoke et al., 2026).

CONCLUSION

The reviewed evidence supported an association between diabetes and regional fascial remodeling, particularly plantar fascia thickening. Glycation and extracellular matrix modification provided a plausible biological link to altered mechanics and reduced function, fulfilling the review's aim of connecting mechanisms with clinical observations. However, heterogeneous mechanical findings and limited causal evidence supported diabetic fasciopathy as an emerging framework rather than a validated independent diagnosis. Current care should prioritize metabolic management, mobility, treatment of regional disorders, and established diabetic foot prevention. Prospective validation is needed before fascial imaging or targeted therapies can guide routine clinical decisions.

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