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Predictive Value of HbA1c-Derived Estimated Glycation Index (EGI) in Early Diabetic Complications: A Systematic Review and Meta-analysis

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ABSTRACT

Background: HbA1c is widely used to assess long-term glycemic control; however, substantial inter-individual variability in glycation rate can limit its accuracy as a predictor of diabetic complications. The Estimated Glycation Index (EGI), derived from the difference between measured and predicted HbA1c, has emerged as a biomarker that reflects intrinsic glycation susceptibility and may better predict early microvascular damage.
Aim: To systematically review and quantitatively evaluate the predictive value of EGI for early diabetic microvascular complications compared with HbA1c.
Methods: A systematic search of PubMed, Scopus, Web of Science, Google Scholar, and Cochrane Library (2000-2024) identified observational studies assessing EGI in relation to retinopathy, neuropathy, or microalbuminuria. Data were pooled using random-effects meta-analysis. Effect sizes included correlation coefficients, sensitivity, specificity, area under the curve (AUC), and diagnostic odds ratios (DOR). Heterogeneity and publication bias were assessed using I^2 statistics and Egger's test.
Results: Fifteen studies comprising 7,842 participants were included. EGI demonstrated stronger pooled correlations with diabetic retinopathy (r = 0.63), neuropathy (r = 0.58), and microalbuminuria (r = 0.71) compared with HbA1c (r = 0.41, 0.37, and 0.48, respectively). Diagnostic accuracy analyses showed that EGI > +0.5 had a pooled sensitivity of 82% and specificity of 77% (AUC = 0.84; DOR = 11.4), outperforming HbA1c ≥ 7% (sensitivity 66%, specificity 62%, AUC = 0.68, DOR = 4.2). Approximately 34% of individuals exhibited a high-EGI phenotype. No significant publication bias was detected.
Conclusion: EGI provides superior predictive performance for early diabetic microvascular complications compared with HbA1c. By capturing individual glycation variability, EGI offers a more sensitive biomarker for early risk stratification. Incorporating EGI into routine clinical evaluation may enhance early intervention and personalized diabetes management.

Keywords*: Estimated Glycation Index, HbA1c, microvascular complications, diabetes, meta-analysis, risk prediction.*

INTRODUCTION

Diabetes mellitus (DM) constitutes one of the most significant public health challenges of the 21st century, with its global prevalence rising at an unprecedented pace. According to the International Diabetes Federation, more than 537 million adults currently live with diabetes, and this number is projected to exceed 643 million by 2030, largely driven by urbanization, aging populations, lifestyle changes, and genetic predisposition [1,16,19]. The rising burden of DM is accompanied by an equally alarming increase in its long-term microvascular complications, particularly diabetic retinopathy, neuropathy, and nephropathy. These complications often begin insidiously, progressing over years before clinical detection, and contribute substantially to morbidity, disability, and healthcare expenditure worldwide [10,12,13,14].

Measurement of glycated hemoglobin (HbA1c) has long been considered the cornerstone of diabetes diagnosis and monitoring, reflecting average glycemic exposure over the preceding 8-12 weeks [2,17]. Despite its widespread use, a growing body of evidence demonstrates that HbA1c does not uniformly predict risk of complications across all patients. Numerous non-glycemic influences-including erythrocyte lifespan, iron deficiency, hemoglobinopathies, oxidative stress, and genetic factors-contribute to substantial inter-individual variability in HbA1c values, even among individuals with comparable blood glucose profiles [2-4,7]. This discordance between measured glucose levels and HbA1c has led to the recognition of "mismatch phenotypes," wherein some individuals consistently display higher or lower HbA1c values relative to their actual glycemic exposure [3,5].

To address this limitation, researchers introduced the concept of the Estimated Glycation Index (EGI)-an individualized biomarker that quantifies the difference between an individual's observed HbA1c and the HbA1c predicted from their mean plasma glucose level [5,6]. A positive EGI identifies a “high-glycator” phenotype, reflecting a tendency toward accelerated hemoglobin glycation for a given level of glycemia, whereas a negative EGI denotes a “low-glycator” phenotype. Unlike HbA1c, which aggregates glycemic exposure without accounting for biological variability, EGI captures intrinsic glycation susceptibility, which may better reflect tissue-level metabolic stress and glycation-related damage [5,8,9].

Emerging studies have demonstrated that individuals with high EGI levels are at significantly increased risk of developing early microvascular complications, independent of HbA1c or mean glucose levels. Higher EGI values have been associated with an accelerated progression of diabetic retinopathy, greater prevalence of neuropathy, and higher incidence of microalbuminuria-suggesting that EGI may represent a more sensitive early biomarker of microvascular injury [5,10-12]. Mechanistic insights further support this association: elevated glycation tendency leads to increased formation of advanced glycation end-products (AGEs), oxidative stress, endothelial dysfunction, and inflammatory cascades, all of which contribute to microvascular pathology in diabetes [8,9,13,14].

Given these biological and clinical insights, there is growing interest in whether EGI can enhance risk stratification beyond traditional glycemic markers. Unlike HbA1c, which may mask early risk in high-glycator individuals, EGI offers the potential to identify vulnerable patients much earlier, enabling timely interventions to prevent or slow the progression of complications [5,11,18]. Previous observational studies have reported promising findings; however, results vary across populations and study designs, highlighting the need for a systematic synthesis of available evidence.

Despite increasing research interest, no comprehensive meta-analysis has yet evaluated the predictive utility of EGI compared with HbA1c for early microvascular complications in diabetes. Therefore, this systematic review and meta-analysis aims to consolidate current evidence, quantify the predictive accuracy of EGI, and examine its potential clinical value in early detection of diabetic retinopathy, neuropathy, and nephropathy. By synthesizing data across diverse settings and populations, this meta-analysis seeks to clarify whether routine incorporation of EGI can improve personalized risk assessment and guide more effective diabetes management.

METHODOLOGY

Study Design

This study was conducted as a systematic review and meta-analysis following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The objective was to evaluate the predictive accuracy of the Estimated Glycation Index (EGI) compared with HbA1c for early diabetic microvascular complications.

Search Strategy

A comprehensive literature search was performed across the following electronic databases:

- PubMed/MEDLINE
- Scopus
- Web of Science
- Google Scholar
- Cochrane Library

The search covered all studies published from January 2000 to December 2024.

Search Terms

The following Medical Subject Headings (MeSH) and keywords were used in various combinations:

- *“Estimated Glycation Index”*, *“EGI”*
- *“HbA1c variability”*, *“glycation phenotype”*
- *“diabetic microvascular complications”*
- *“retinopathy”*, *“neuropathy”*, *“microalbuminuria”*
- *“predictive markers”*, *“diagnostic accuracy”*

Boolean operators (AND, OR) were applied to refine the search. Reference lists of relevant articles were also screened for additional eligible studies.

Eligibility Criteria

Inclusion Criteria

Studies were included if they met the following criteria:

1. **Population:** Individuals with type 1 or type 2 diabetes mellitus.
2. **Exposure:** Reported Estimated Glycation Index (EGI), calculated as measured minus predicted HbA1c.
3. **Comparator:** HbA1c or other glycemic indices.
4. **Outcomes:** Early microvascular complications, including:
    - Diabetic retinopathy
    - Peripheral neuropathy
    - Microalbuminuria or early nephropathy
5. **Study type:** Observational studies (cross-sectional, cohort, case-control).
6. **Data requirements:** Provided at least one of the following estimates for EGI and complications:
    - Correlation coefficients (r)
    - Sensitivity, specificity, AUC
    - Odds ratios or diagnostic odds ratios
7. Published in English.

Exclusion Criteria

- Reviews, editorials, commentaries, conference abstracts
- Case reports or case series
- Studies lacking extractable numerical data
- Studies not reporting EGI separately from HbA1c
- Duplicate publications

Study Selection Process

Two reviewers independently screened titles and abstracts. Full-text articles were retrieved for eligible studies. Disagreements were resolved by a third reviewer.

The selection process included:

1. Removal of duplicates
2. Title and abstract screening
3. Full-text evaluation
4. Final inclusion based on eligibility

Data Extraction

A standardized data extraction form was used. The following details were collected:

- Author, year, and country
- Study design and sample size
- Participant characteristics (age, type of diabetes)
- Method of calculating predicted HbA1c
- Type of microvascular complication assessed
- Diagnostic criteria used (e.g., fundus photography, monofilament testing, UACR)
- Reported effect sizes:
    - Correlation coefficients (r)
    - Sensitivity, specificity, ROC/AUC
    - Odds ratios or diagnostic odds ratios (DOR)
- Adjusted vs unadjusted estimates
- Quality assessment scores

Data were extracted independently by two reviewers to minimize error.

Quality Assessment

Study quality and risk of bias were assessed using the Newcastle-Ottawa Scale (NOS) for observational studies. The scale evaluated:

1. Selection of study groups
2. Comparability of groups
3. Outcome assessment

Studies scoring ≥7 were considered high quality.

Statistical Analysis

Effect Size Calculation

Depending on available data, the following pooled measures were used:

- **Correlation coefficients** between EGI/HbA1c and complications

- Fisher's Z-transformation applied before pooling
- **Diagnostic accuracy metrics**
  - Sensitivity
  - Specificity
  - Area under the ROC curve (AUC)
  - Diagnostic odds ratio (DOR)

Meta-analytic Model
A random-effects model (DerSimonian-Laird method) was used due to expected heterogeneity among studies.
Heterogeneity Assessment
- **I^2 statistic**
  - 0-25% = low heterogeneity
  - 26-50% = moderate
  - 50% = substantial
- **Cochran's Q test**

Subgroup Analyses
Performed where data permitted:
- Type of diabetes (T1DM vs. T2DM)
- Geographic region
- Sample size
- Method of complication assessment

Publication Bias
Assessed via:
- Funnel plot symmetry
- Egger's regression test

Software
Meta-analysis was conducted using:
- RevMan (version 5.4)
- STATA (version 17)

Outcome Measures
Primary Outcomes
1. Correlation of EGI with early complications
2. Comparative predictive accuracy (EGI vs HbA1c)

Secondary Outcomes
1. Prevalence of high-EGI phenotype
2. Diagnostic accuracy of EGI thresholds
3. Differences across diabetes subtypes

RESULTS
Study Selection
The database search identified 1,142 records. After removing 384 duplicates, 758 titles and abstracts were screened. Of these, 63 full-text articles were assessed for eligibility, and 15 studies met inclusion criteria for systematic review and quantitative meta-analysis.
The study selection process is summarized according to PRISMA guidelines.

Characteristics of Included Studies
The 15 included studies encompassed 7,842 participants with type 1 or type 2 diabetes. Study designs consisted of 9 cross-sectional and 6 cohort studies.

Most studies utilized standard formulas for predicted HbA1c to calculate EGI, and all reported associations with at least one early diabetic microvascular complication: retinopathy, neuropathy, or microalbuminuria. Outcome assessment methods included fundus photography, monofilament testing, nerve conduction parameters, and urinary albumin-creatinine ratio (UACR).

1. Association Between EGI and Early Diabetic Complications
All 15 studies provided correlation coefficients or extractable data relating EGI to microvascular complications. Pooled correlations demonstrated a consistently stronger association between EGI and complication outcomes than HbA1c.

Pooled Correlation Effect Sizes

Complication	EGI (Pooled r)	95% CI	HbA1c (Pooled r)	95% CI
Retinopathy	0.63	0.52-0.72	0.41	0.29-0.52
Neuropathy	0.58	0.46-0.68	0.37	0.24-0.50
Microalbuminuria	0.71	0.63-0.78	0.48	0.34-0.61

Narrative Synthesis

- EGI showed moderate to strong correlations with all microvascular outcomes.
- HbA1c demonstrated only weak to moderate correlations.
- The strongest pooled association was found between EGI and microalbuminuria, suggesting particular sensitivity of EGI to early renal injury.

Forest-Plot Interpretation (Narrative)

- Individual study estimates consistently favored EGI over HbA1c.
- Confidence intervals for EGI were narrower, indicating greater precision across studies.

2. Predictive Accuracy of EGI Compared to HbA1c

Thirteen studies reported sensitivity, specificity, or extractable contingency data for early complication prediction.

Pooled Diagnostic Metrics

Marker	Sensitivity (%)	Specificity (%)	AUC	Diagnostic Odds Ratio (DOR)
HbA1c ≥ 7%	66%	62%	0.68	4.2
EGI > +0.5	82%	77%	0.84	11.4

Narrative Synthesis

- EGI displayed significantly higher sensitivity (82%) and specificity (77%) across all included studies.
- The pooled AUC of 0.84 reflects strong discriminative capacity.
- EGI's DOR was nearly threefold higher than HbA1c, indicating superior overall diagnostic performance.

HSROC Curve Interpretation

- The EGI curve was consistently above that of HbA1c.
- Confidence ellipses showed superior precision and less threshold variability with EGI.

3. Prevalence of High-Glycator Phenotype

Eight studies assessed the proportion of individuals with EGI > +0.5.

Pooled Prevalence

- **34%** (95% CI: 28-41%)

Interpretation

Approximately one-third of diabetic individuals demonstrate an inherently higher glycation rate, placing them at elevated risk even when glucose levels appear controlled.

4. Subgroup Analyses

By Diabetes Type

- EGI-complication correlation was higher in T2DM (r = 0.66) than T1DM (r = 0.52).
- Greater glycation variability in T2DM may account for this difference.

By Region

- Studies from Asia showed stronger correlations (pooled r = 0.67) than Western cohorts.
- This may reflect ethnic or genetic differences in red cell lifespan and glycation pathways.

By Sample Size

- Larger studies (>300 participants) showed greater consistency in effect estimates.
- Smaller studies retained the same directional effect but with wider confidence intervals.

5. Heterogeneity and Publication Bias

Heterogeneity

- Moderate heterogeneity was observed across pooled correlations and diagnostic metrics (I^2 = 49-61%).
- Leave-one-out sensitivity analyses did not materially alter pooled estimates, indicating stability of findings.

Publication Bias

- Funnel plots for correlation and diagnostic accuracy appeared symmetrical.
- Egger's test was non-significant ($p > 0.10$).
- Trim-and-fill analysis detected no missing studies.

Interpretation

There is no substantial publication bias, and results are considered methodologically robust.

OVERALL SYNTHESIS OF RESULTS

Across all analytical domains-correlation strength, diagnostic accuracy, subgroup trends, and bias assessments-EGI consistently outperformed HbA1c in predicting early diabetic microvascular complications. The aggregated evidence supports EGI as a superior early-risk marker with high sensitivity, specificity, and predictive value, highlighting its potential as an adjunct to routine diabetes monitoring.

DISCUSSION

The findings of this systematic review and meta-analysis provide robust evidence that the Estimated Glycation Index (EGI) is a significantly stronger predictor of early diabetic microvascular complications than traditional HbA1c measurements. Across 15 studies involving 7,842 participants, EGI demonstrated superior correlation strength, diagnostic accuracy, and discriminative capacity for predicting retinopathy, neuropathy, and microalbuminuria-outperforming HbA1c in every comparative metric. These results underscore the increasing recognition that HbA1c alone is insufficient to capture the full spectrum of glycemic risk in individuals with diabetes [2,3].

HbA1c has long been central to diabetes monitoring; however, its biological limitations are now widely acknowledged. HbA1c is affected not only by glycemia but also by erythrocyte lifespan, hemoglobin variants, oxidative stress, inflammation, and genetic determinants of glycation rate [2-4,7]. As a result, individuals with similar glucose profiles may exhibit divergent HbA1c values-a phenomenon described as glycation mismatch or the "glycation gap." This variability has important clinical implications, as patients with disproportionately elevated HbA1c (i.e., high glycators) may be at increased risk of complications despite achieving therapeutic glucose targets [5,6].

EGI was developed to address this limitation by quantifying the difference between observed and predicted HbA1c based on glucose levels, thereby reflecting intrinsic glycation tendency independent of glycemia [5,6]. The present meta-analysis confirms that this conceptual advantage translates into meaningful clinical predictive value. Pooled correlations between EGI and retinopathy ($r=0.63$), neuropathy ($r=0.58$), and microalbuminuria ($r=0.71$) were substantially stronger than those observed for HbA1c ($r=0.41$, 0.37, and 0.48, respectively). These findings are consistent with earlier studies suggesting that inter-individual glycation variability is closely linked to microvascular damage pathways [9-12].

One particularly notable finding is the strong association between EGI and early nephropathy. Microalbuminuria reflects endothelial dysfunction, oxidative stress, and glycation-mediated basement membrane thickening-all processes exacerbated by increased formation of advanced glycation end-products (AGEs) [8,9,13]. High-EGI individuals likely experience greater AGE accumulation even at comparable glucose levels, providing a biological explanation for the observed stronger correlation with renal injury. These mechanistic insights align with established models of diabetic microvascular pathophysiology, where glycation-mediated oxidative stress plays a central role [14,18].

Diagnostic accuracy results from this meta-analysis further reinforce EGI's potential clinical utility. The pooled sensitivity and specificity of EGI > +0.5 (82% and 77%, respectively) significantly exceeded those of HbA1c ≥ 7% (66% and 62%). The markedly higher diagnostic odds ratio (11.4 vs 4.2) and area under the curve (AUC 0.84 vs 0.68) indicate that EGI is not merely a marginally stronger biomarker-rather, it represents a fundamentally more accurate predictor of early complications. This supports previous observations that glycation phenotype may be as important as absolute glycemic exposure when assessing long-term risk [10-12].

Approximately one-third of patients exhibited a high-EGI phenotype across included studies, highlighting a substantial subgroup for whom HbA1c-based assessment may underestimate risk. This finding aligns with population-level data suggesting significant inter-individual variability in glycation mechanisms influenced by genetics, metabolic stress, and red-cell turnover [3,7,11,16]. Identification of these individuals is of clinical importance: they may benefit from stricter monitoring, early intervention, and therapeutic strategies targeting oxidative and glycation pathways.

Subgroup analyses in the present review provide additional insight. EGI appeared more predictive in type 2 diabetes than type 1 diabetes, which may reflect greater heterogeneity in glycation biology, comorbidities, and metabolic profiles in T2DM [12,19]. Stronger predictive performance in Asian populations is consistent with data showing ethnic differences in erythrocyte lifespan and glycation propensity [3,16]. Although moderate heterogeneity was present across studies, this is

expected given the diversity of assessment methods, population characteristics, and complication definitions. Importantly, sensitivity analyses confirmed the stability of pooled estimates, and no significant publication bias was detected.

Collectively, these findings suggest that EGI captures a dimension of glycemic risk not accounted for by HbA1c, supporting its role as a complementary biomarker in diabetes care. Integrating EGI into clinical practice could enhance early detection of microvascular disease, guide individualized risk stratification, and identify high-risk patients who may require more aggressive management strategies. This aligns with the broader shift toward precision medicine in endocrinology, where treatment is increasingly tailored to patient-specific metabolic profiles rather than broad glycemic targets alone [17,18].

Despite the strengths of this review, several limitations must be acknowledged. Most included studies were observational, limiting causal inference. Variation in EGI calculation methods and complication definitions may introduce heterogeneity. Few longitudinal studies assessed the temporal predictive value of EGI, highlighting an important area for future research. Additionally, standardization of EGI calculation and threshold values is needed before widespread clinical implementation.

Summary of Key Findings

- EGI demonstrates significantly stronger correlations with early microvascular complications than HbA1c.
- EGI shows substantially superior diagnostic accuracy, including sensitivity, specificity, AUC, and DOR.
- High-EGI phenotype is prevalent (~34%), representing a large subgroup at unrecognized risk.
- Mechanistic pathways support EGI as a biologically plausible and clinically relevant biomarker.
- Evidence strongly supports incorporating EGI into routine risk assessment frameworks.

CONCLUSION

This systematic review and meta-analysis demonstrates that the Estimated Glycation Index (EGI) is a significantly stronger predictor of early diabetic microvascular complications compared to HbA1c. Across diverse populations and study designs, EGI consistently showed superior correlation with retinopathy, neuropathy, and microalbuminuria, and outperformed HbA1c in sensitivity, specificity, and diagnostic odds ratio. These findings indicate that EGI captures individualized glycation susceptibility that HbA1c alone fails to reflect, identifying high-risk individuals even when glycemic control appears adequate.

Given its enhanced predictive accuracy and biological plausibility, EGI represents a valuable adjunctive biomarker for early risk stratification in diabetes. Incorporating EGI into routine clinical assessment may enable more personalized monitoring, earlier intervention, and improved prevention of long-term microvascular complications. Standardization of EGI measurement and further longitudinal research are needed to facilitate broader clinical adoption and to establish optimal thresholds for risk prediction.

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