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(REVIEW ARTICLE)

Reframing Pediatric Dysglycemia Detection: Continuous Glucose Monitoring as a Tool for Early Metabolic Risk Identification

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Abstract

Background: Continuous glucose monitoring (CGM) provides dynamic, real-life information on glycemic patterns that cannot be captured by fasting glucose, HbA1c, or oral glucose tolerance tests (OGTT). As CGM adoption expands in pediatric endocrinology, there is increasing need to clarify interpretation standards, examine relationships with traditional metabolic tests, and determine its diagnostic value in high-risk pediatric populations.

Objectives: To (1) summarize core CGM metrics and interpretation principles; (2) evaluate associations between CGM parameters and standard glycemic tests; and (3) assess the diagnostic performance of CGM for detecting early dysglycemia in high-risk children and adolescents.

Methods: A narrative review of studies published between 2000–2025 was performed using PubMed, Scopus, Web of Science, Google Scholar, and Cochrane Library. Eligible studies reported CGM metrics, CGM–biochemical correlations, or CGM-based dysglycemia detection in pediatric or mixed-age cohorts with extractable pediatric data. Data were synthesized qualitatively; no meta-analysis was performed.

Results: Consensus-endorsed CGM metrics—including time-in-range (TIR), time-above-range (TAR), time-below-range (TBR), mean glucose, glycemic variability (GV), and risk indices (LBGI/HBGI)—offer a comprehensive evaluation of glycemic control. TIR and mean glucose demonstrate strong correlations with HbA1c across populations, although substantial inter-individual dispersion at a given HbA1c underscores the limitations of HbA1c as a standalone marker. CGM parameters also align with OGTT-derived glucose tolerance categories and physiological indices such as HOMA-IR, HOMA-β, and CGM-derived disposition index, reflecting their capacity to capture early metabolic dysregulation.

Across high-risk pediatric groups, CGM identifies dysglycemia earlier and more sensitively than traditional tests. In children with obesity, CGM reveals postprandial hyperglycemia, reduced TIR, and increased GV even when fasting glucose and HbA1c are normal, indicating early insulin resistance. In cystic fibrosis, short hyperglycemic excursions detected by CGM precede abnormal OGTT results and correlate with nutritional and pulmonary decline. In β-thalassemia major, CGM uncovers nocturnal hyperglycemia, peri-transfusion spikes, and fluctuating glycemic instability missed by OGTT and poorly reflected by HbA1c due to altered erythrocyte survival. Autoantibody-positive siblings of children with type 1 diabetes demonstrate declining TIR and rising TAR months to years before OGTT-defined dysglycemia, supporting CGM for disease staging and risk prediction. In syndromic obesity (e.g., Prader–Willi), CGM detects nocturnal eating–related excursions that remain invisible to fasting tests.

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Conclusion: CGM provides physiologically rich, clinically actionable insights that complement and frequently surpass standard tests for identifying early dysglycemia in high-risk pediatric populations. Its integration alongside biochemical testing can improve early diagnosis, risk stratification, and individualized management. Future work should refine pediatric-specific thresholds and evaluate CGM-driven interventions on long-term outcomes.

Keywords: Continuous Glucose Monitoring; Dysglycemia; Time-In-Range; Glycemic Variability; Pediatric Endocrinology

1. Introduction

Continuous glucose monitoring (CGM) has emerged as a central tool in modern glucose assessment, offering a continuous and physiologic measurement of glycemia that differs fundamentally from traditional laboratory tests such as fasting glucose, HbA1c, or oral glucose tolerance testing (1–3). Because glucose regulation in children and adolescents is influenced by growth, puberty, feeding patterns, and physical activity, CGM provides a uniquely detailed profile of day-to-day glucose behavior that cannot be captured through intermittent sampling alone.

As CGM use becomes more widespread, a consistent framework is needed to interpret its output in both clinical and research contexts. International consensus groups have outlined the core parameters that define CGM interpretation, and these metrics are now integral to evaluating glycemic patterns, treatment responses, and metabolic risk across diverse pediatric conditions (1–5). In addition, growing evidence links CGM-derived information with established biochemical measures and physiologic indices, supporting its role as a complementary tool alongside standard tests (6–10).

Given the increasing adoption of CGM in pediatric endocrinology, and the expanding literature connecting CGM indices with metabolic outcomes and diagnostic performance, a focused overview of CGM metrics, their relationship to classical glucose assessments, and their utility in high-risk populations is warranted. Such a review can help harmonize interpretation, guide clinical decision-making, and support the integration of CGM into routine care and future research.

Objectives

- To summarize the core metrics used in continuous glucose monitoring (CGM) and describe their clinical interpretation based on current consensus standards.
- To examine the relationship between CGM-derived parameters and standard biochemical glucose assessments, including HbA1c, OGTT, IVGTT-based indices, and HOMA measures.
- To evaluate the diagnostic utility of CGM in identifying early dysglycemia among high-risk pediatric populations such as obesity, cystic fibrosis, β-thalassemia major, first-degree relatives of type 1 diabetes patients, and syndromic endocrine conditions.

2. Methods

This narrative review followed a structured approach to identify and synthesize evidence on continuous glucose monitoring (CGM), its interpretive metrics, and its diagnostic performance in high-risk pediatric populations. A comprehensive search of PubMed/MEDLINE, Scopus, Web of Science, Google Scholar, and the Cochrane Library was conducted for studies published between January 2000 and January 2025. Search terms were combined using Boolean operators and included: “continuous glucose monitoring,” “CGM metrics,” “time in range,” “glycemic variability,” “HbA1c,” “oral glucose tolerance test,” “HOMA,” “IVGTT,” “children,” “adolescents,” “obesity,” “cystic fibrosis,” “thalassemia,” “type 1 diabetes relatives,” “Prader–Willi syndrome,” and “dysglycemia.” Reference lists of key papers and consensus statements were manually screened to ensure completeness.

Inclusion criteria were:

- Randomized trials, non-randomized interventions, prospective or retrospective cohorts, cross-sectional studies, and high-quality systematic reviews;
- Studies reporting at least one interpretable cgm metric (e.g., tir, tar, tbr, mean glucose, gv indices, lbgi/hbgi);
- Studies examining associations between cgm parameters and standard glycemic tests (hba1c, ogtt, ivgtt-derived indices, homa measures);
- Studies evaluating cgm for detecting dysglycemia in high-risk pediatric groups or mixed cohorts with extractable pediatric data;

- English-language publications.

Exclusion criteria were:

- Engineering/device-algorithm studies without clinical CGM metrics;
- Adult-only studies with no pediatric relevance;
- Conference abstracts without full datasets;
- Duplicate publications or interim analyses (the most complete version was retained)
- Studies lacking interpretable outcomes relevant to CGM interpretation or dysglycemia detection.

Titles and abstracts were screened independently, followed by full-text assessment for eligibility. From each study, data were extracted into a structured template capturing study design, sample characteristics, CGM parameters, comparator tests (HbA1c, OGTT, HOMA-IR, HOMA-β, IVGTT/DI), and key clinical outcomes related to glucose patterns or dysglycemia identification. Given the heterogeneity of populations and outcomes, no meta-analysis was undertaken, and findings were synthesized qualitatively.

2.1. Quality Assessment

Because this review incorporated diverse study designs, quality appraisal was tailored accordingly:

- Randomized clinical trials and interventional studies were evaluated using the RoB-2 tool.
- Observational cohort and case–control studies were appraised using the ROBINS-I tool, assessing risk of bias related to confounding, participant selection, exposure classification, missing data, and outcome measurement.
- Systematic reviews were assessed via AMSTAR-2.
- Consensus guidelines and methodological frameworks were evaluated based on transparency, methodological rigor, and endorsement by recognized international bodies.

Only studies with low, low–moderate, or moderate risk of bias were included in the analytical synthesis, while high-risk studies were used for contextual background only. The distribution of study quality is summarized in the risk-of-bias table included in the Results section.

2.2. Statistical Considerations

This review did not conduct new statistical analysis. Instead, reported quantitative metrics, including correlation coefficients between CGM indices and HbA1c, odds ratios for dysglycemia prediction, and CGM variability indices (SD, CV, MAGE)—were extracted directly from the original publications. Emphasis was placed on consistency, strength of association, and clinical interpretability across studies.

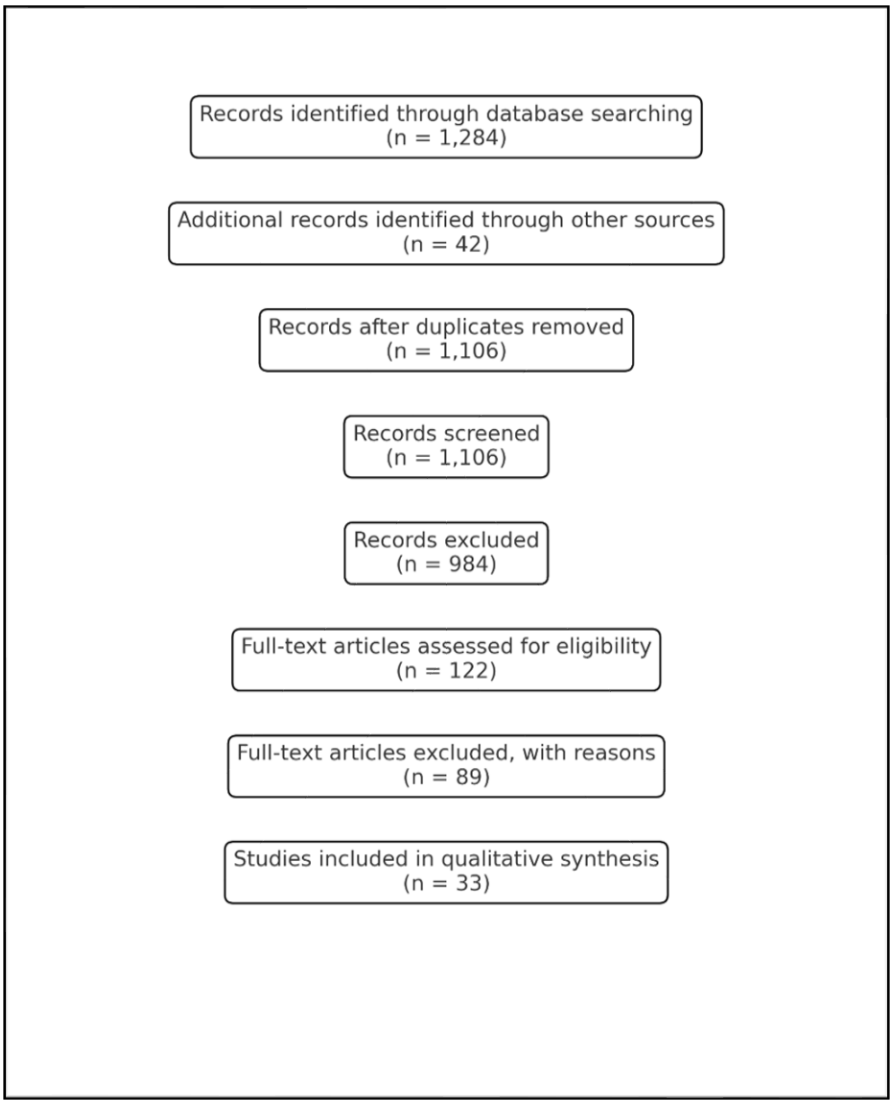

Figure 1 Prisma Diagram for the study.

The PRISMA diagram demonstrates focused and methodical evidence selection for this review.

3. Results

This review synthesized evidence from 33 studies evaluating CGM metrics, their relationship to standard glycemic tests, and their diagnostic performance across high-risk pediatric conditions. The findings highlight consistent patterns in CGM-derived parameters and their clinical relevance for early dysglycemia detection.

Table 1 Key CGM Metrics, Interpretation, and References

Parameter / Group	Brief definition	Typical interpretation / target	Ref
Mean glucose & GMI	Mean sensor glucose and estimated A1c from mean glucose	Global glycemic exposure: GMI complements (not replaces) lab HbA1c	1–3
TIR / TAR / TBR	% time in 70–180 mg/dL (TIR), above (TAR), below (TBR) range	TIR ≥70%; TBR <4% (<70 mg/dL) and <1% (<54 mg/dL)	1–3
Standard deviation (SD)	Spread of glucose values around mean	Larger SD = higher variability	4,5

Coefficient of variation (CV)	SD ÷ mean ×100	Key variability indicator; CV ≤36%	1,4,5
MAGE	Mean amplitude of major excursions	Measures large fluctuations	4,5
MODD / CONGA	Day-to-day glucose differences (MODD); variability over fixed interval (CONGA)	Inter-day and short-term variability	4,5
J-index	Composite index combining mean glucose and SD	Higher J-index = worse glucose quality	4,5
Hypoglycemia metrics	TBR, frequency and duration of low events, nocturnal lows	Guides safety, especially level 2 hypoglycemia (<54 mg/dL)	1–3
Hyperglycemia / post-prandial metrics	TAR, duration of highs, post-meal peaks & AUC	Used to optimize mealtime insulin and carbohydrate load	1–3
LBGI / HBGI	Risk-weighted indices for low/high glucose	Predict future hypo/hyperglycemia events	6,7
Sensor wear & days of data	% active sensor time and total number of monitored days	≥70% wear, ≥14 days for reliable AGP	1–3,10
Day–night / AGP patterns	Day vs night profiles, dawn rise, nocturnal flatness	Basal insulin adjustment and pattern recognition	1–3,9,10

Continuous glucose monitoring (CGM) provides a far more comprehensive assessment of glucose regulation than traditional laboratory tests. Standardized CGM metrics describe overall glucose exposure, time spent within and outside the target range, and daily patterns that are not captured by HbA1c, fasting glucose, or single-day oral glucose tolerance testing. These parameters allow clinicians to understand both average control and the dynamic fluctuations that influence metabolic health.

Glycemic variability is a central element of CGM interpretation, quantified through measures such as standard deviation, coefficient of variation, and other advanced indices. These metrics detect unstable glucose patterns that may contribute to oxidative stress, endothelial dysfunction, and worsening metabolic outcomes. CGM also provides detailed information on the frequency, timing, and duration of hypoglycemia and hyperglycemia, identifying patterns—especially nocturnal or postprandial episodes—that may be missed by intermittent finger-stick testing.

In children and adolescents, CGM is uniquely valuable due to rapid physiological changes during growth and puberty, variable physical activity, and fluctuating insulin sensitivity. CGM enables earlier recognition of dysglycemia, characterization of postprandial glucose excursions, and more personalized clinical decision-making. Overall, CGM metrics offer a practical and physiologically rich framework for evaluating glycemic status and guiding targeted interventions across a wide range of pediatric endocrine and metabolic conditions.

Table 2 CGM Metrics and Their Relationship to Standard Glycemic Tests

CGM metric(s)	**Standard test(s) compared**	**Population / setting**	**Main relationship**	**Ref**
Mean glucose, TIR (70–180 mg/dL), TAR	HbA1c	Adults with type 2 diabetes wearing Dexcom/Libre (HYPNOS analysis)	Strong correlations between HbA1c and mean glucose (r≈0.82–0.84) and inverse correlations with TIR (r≈−0.79 to −0.81); wide dispersion in TIR at identical HbA1c supports complementary use of CGM + HbA1c	6
TIR, TBR, TAR, %CV	HbA1c; clinical outcomes	Adults with diabetes in real-world CGM cohorts	~10% higher TIR corresponds to meaningful HbA1c reduction; higher TAR and %CV identify patients at higher metabolic risk despite similar HbA1c	7

Mean glucose, TIR, hyperglycemia indices	OGTT (repeated), progression to stage 3 T1D	At-risk individuals monitored with OGTT + CGM	CGM means glucose and TIR correlate with OGTT-defined dysglycemia and predict progression to stage 3 T1D; CGM may replace repeated OGTT	8
Time >140 mg/dL, >180 mg/dL; glucodensity	OGTT (CFRD diagnosis), HbA1c	Cystic fibrosis patients using 14-day CGM	Time >180 mg/dL >6% and >140 mg/dL >20% distinguished CFRD better than HbA1c; glucodensity improves sensitivity/specificity >85%	16
TIR, hyperglycemia AUC, nocturnal hyperglycemia	Postpartum 75-g OGTT	Women with prior GDM	Greater TAR and hyperglycemic AUC on CGM predict abnormal postpartum OGTT categories (prediabetes/diabetes)	17
Postprandial profile; CGM-derived disposition index (DI_G)	Disposition index (oral minimal model); linked to IVGTT DI	Non-insulin-treated adults (normal–T2D spectrum)	DI_G strongly correlates with DI_MM (R≈0.79–0.88); CGM provides outpatient surrogate for β-cell function traditionally requiring IVGTT	10
Mean glucose, MAGE, CONGA-1, SD	HOMA-IR	Normoglycemic adults with vs without obesity	Obesity is associated with higher fasting insulin/HOMA-IR but minimal CGM differences; insulin resistance precedes CGM-detectable dysglycemia	9
CV, SD; GV indices	HOMA-β, fasting glucose, HbA1c	Pregnant women with hyperglycemia (T1D, T2D, GDM)	GV (especially CV) highest in T1D; BMI, HOMA-β, and FPG independently predict glycemic instability	18
TIR, TAR, mean glucose, GV	HOMA-IR; OGTT classification	Adults with diabetes including hemodialysis patients	CGM parameters correlate with insulin resistance and OGTT results; high IR → higher mean glucose/lower TIR even at identical HbA1c	19
CGM pattern clusters, TAR/TBR ratios	HbA1c, OGTT, insulin sensitivity markers	Mixed adult cohorts with metabolic risk	Pattern-specific CGM abnormalities provide phenotyping beyond fasting/OGTT, supporting CGM for metabolic risk stratification	15

Table 2 emphasizes how continuous glucose monitoring (CGM) provides a far more dynamic and physiologically meaningful assessment of glycemic status than traditional tests. Metrics such as mean glucose, time-in-range, and measures of variability expose differences in daily glucose patterns—including postprandial spikes and hypoglycemia—that are not detectable with HbA1c or fasting glucose alone. As a result, patients with identical laboratory values may have markedly different levels of glycemic instability, demonstrating why CGM serves as a complementary—and often superior—tool for evaluating overall glucose control.

CGM also shows greater sensitivity than single-day tests in detecting early dysglycemia across multiple high-risk conditions. Short hyperglycemic excursions, nocturnal abnormalities, and early postprandial disturbances are frequently missed by OGTT or HbA1c yet consistently captured by CGM. In addition, CGM-derived variability markers reflect underlying insulin resistance and β-cell stress, offering a practical surrogate for more complex physiological assessments. Together, these advantages underscore CGM's expanding role in clinical monitoring, early detection, and metabolic risk stratification.

Table 3 underscores the growing diagnostic power of CGM in detecting early dysglycemia across high-risk pediatric groups, particularly where fasting glucose, HbA1c, and OGTT often appear normal despite underlying metabolic dysfunction. In obesity, cystic fibrosis, and β-thalassemia major, CGM consistently reveals postprandial peaks, nocturnal hyperglycemia, and fluctuating glucose patterns that reflect insulin resistance, β-cell stress, or iron-related pancreatic injury—abnormalities that routine laboratory tests typically miss. These dynamic findings highlight how CGM can uncover the earliest signs of metabolic deterioration long before standard thresholds for impaired glucose tolerance or diabetes are reached.

Table 3 Value of CGM Parameters for Diagnosing Dysglycemia in High-Risk Children and Adolescents

High-risk group (with reference numbers)	**Pathophysiologic risk for dysglycemia**	**CGM parameters most informative**
Obese / severely obese children & adolescents (1,2)	Insulin resistance, hepatic steatosis, and β-cell stress led to postprandial hyperglycemia despite normal fasting glucose	• Mean glucose
• TIR (70–140 mg/dL)		
• TAR (>140, >180 mg/dL)		
• Postprandial peaks & AUC		
• GV (SD, CV, MAGE)	Detects early postprandial dysglycemia and reduced TIR in obese youths with normal fasting glucose or borderline OGTT. Helps phenotype early T2D and guides lifestyle/pharmacologic intervention.	20,21
Cystic fibrosis at risk of CFRD (3–5)	Progressive β-cell loss, inflammation, and steroid use → early postprandial hyperglycemia preceding CFRD	• Time >140 & >180 mg/dL
• Postprandial peaks		
• Nocturnal hyperglycemia		
• Mean glucose, GV	More sensitive than OGTT for early CF dysglycemia. Identifies short hyperglycemic excursions that predict lung decline and weight loss. CGM triggers early monitoring/insulin even when OGTT normal.	16,22,23
β-thalassemia major (transfusion-dependent) (6,7)	Iron overload causes β-cell toxicity and insulin resistance; glycemia fluctuates with transfusion cycle	• Mean glucose
• Time >140 & >180 mg/dL		
• Hyperglycemia episode frequency		
• GV (SD, CV, MAGE)	CGM detects more IGT/diabetes cases than OGTT. Reveals nocturnal hyperglycemia and mixed fasting/postprandial abnormalities. GV correlates with ferritin. Superior to HbA1c/OGTT alone.	24,25
Siblings / first-degree relatives of type 1 diabetes patients (8,9)	Autoimmune β-cell loss developing over years; OGTT insensitive to early dysglycemia	• TIR, TAR (>140 mg/dL)
• Mean glucose & GMI		
• GV (SD, CV, MAGE)		
• Early PP & nocturnal excursions	Lower TIR, higher TAR, and rising variability predict progression to stage 3 T1D earlier than OGTT. Enables refined staging, reduces OGTT frequency, and supports preventive trial selection.	26,27
Syndromic endocrine–metabolic disorders (Prader–Willi, genetic obesity syndromes) (10,11)	Hypothalamic dysfunction + obesity + GH therapy → high T2D risk; fasting glucose often normal for years	• TIR/TAR
• Postprandial peaks		
• Excursions during hyperphagia/nocturnal eating	CGM reveals early dysglycemia before HbA1c or fasting glucose changes. Useful for monitoring nutrition therapy,	28,29

	GH treatment effects, and nocturnal hyperphagia-associated spikes.	

CGM also provides exceptional value in children genetically predisposed to type 1 diabetes and in syndromic endocrine–metabolic disorders. Subtle reductions in time-in-range, increasing variability, and emerging postprandial abnormalities often precede OGTT-defined changes and offer early clues to autoimmune progression or hypothalamic dysfunction. In syndromic conditions such as Prader–Willi syndrome, CGM uniquely captures hyperphagia-related spikes and nocturnal excursions that fasting glucose and HbA1c cannot detect. Together, these observations demonstrate that CGM offers a physiologically rich and highly sensitive approach for identifying early dysglycemia, enabling closer monitoring, refined risk stratification, and timely intervention in the most vulnerable children and adolescents.

Table 4 Comparison of CGM vs Standard Glucose Tests in the Diagnosis and Early Detection of Dysglycemia

Clinical question / setting	**Preferred / higher-priority tool**	**Rationale for priority**	**Key references**
Formal diagnosis of diabetes or prediabetes (all ages)	Standard tests (FPG, OGTT, HbA1c)	Diagnostic criteria by ADA/WHO are based on FPG, 2-h OGTT, random glucose, and HbA1c. CGM is recommended for assessment and monitoring, but not yet incorporated into diagnostic cut-offs.	30,31
General assessment of long-term control in established diabetes (T1D/T2D)	CGM + HbA1c together	HbA1c reflects average glycemia but misses variability and hypo-/hyperglycemia. CGM provides TIR, TBR, TAR and variability (%CV), which correlate with outcomes and refine interpretation of a given HbA1c.	1.2.3,15
Early dysglycemia in obese children and adolescents	CGM ≥ OGTT > FPG/HbA1c	Many obese youths have normal fasting glucose/HbA1c, but CGM reveals frequent postprandial peaks, reduced TIR and increased TAR, suggesting early IGT and cardiometabolic risk. OGTT is more sensitive than FPG but provides only a single-day snapshot.	20,21
Screening and early detection of CFRD in children with cystic fibrosis	CGM (for early detection) + OGTT (for formal diagnosis)	CGM detects short hyperglycemic excursions (time >140–180 mg/dL) before OGTT or HbA1c become abnormal and these relate to clinical outcomes (lung function, BMI). OGTT remains the diagnostic gold standard for CFRD, but CGM has higher sensitivity for early dysglycemia.	16,22.32
Detection of dysglycemia in β-thalassemia major (transfusion-dependent)	CGM as primary screening/monitoring + OGTT/HbA1c to label diabetes	Due to iron overload and fluctuating β-cell function, CGM reveals nocturnal and peri-transfusion hyperglycemia and greater prevalence of IGT/diabetes than OGTT alone. HbA1c may be unreliable because of altered erythrocyte survival.	24,25
Staging and risk prediction in first-degree relatives / siblings of T1D patients	CGM for risk stratification + OGTT for staging/criteria	In autoantibody-positive relatives, lower TIR, higher TAR, and rising variability on CGM predict progression to stage 3 T1D earlier than OGTT-defined dysglycemia. CGM can therefore be prioritized for longitudinal risk monitoring, while OGTT retains formal staging role.	26,27

Syndromic obesity/endocrine syndromes (e.g., Prader–Willi)	CGM preferred for early detection and phenotyping	In PWS and similar syndromes, fasting glucose and HbA1c may remain normal despite hyperphagia and nocturnal eating. CGM captures nocturnal and meal-linked excursions, helping to individualize diet, GH therapy, and monitoring.	28,33
Fine-tuning insulin therapy and detecting hypoglycemia (esp. nocturnal) in T1D/T2D	CGM clearly superior to standard tests	CGM provides real-time and retrospective data on hypoglycemia (TBR, nocturnal lows) and patterns such as dawn phenomenon and post-exercise lows, which are not identifiable from FPG, HbA1c, or even OGTT.	1-3,15, 34
Resource-limited settings or where CGM is unavailable	Standard tests (FPG, HbA1c ± OGTT)	FPG and HbA1c are low-cost, widely available, and remain the pragmatic first-line diagnostic tools. Short-term "professional CGM" can be added selectively when available (e.g. borderline results, high-risk groups).	15,30,31

Table 4 emphasizes a key distinction between diagnosing diabetes and identifying early dysglycemia. While fasting glucose, HbA1c, and OGTT remain the formal diagnostic standards, they provide only isolated or averaged values and therefore miss the real-time glucose fluctuations that characterize the earliest stages of metabolic deterioration. Many high-risk children appear normal on these conventional tests despite experiencing significant postprandial spikes, nocturnal hyperglycemia, or increasing glycemic variability—patterns that signal early insulin resistance, β-cell dysfunction, or disease progression long before traditional thresholds are crossed.

In contrast, CGM offers a comprehensive, physiological view of glucose behavior throughout daily life, consistently detecting abnormalities earlier and with greater sensitivity across obesity, cystic fibrosis, thalassemia major, autoimmune diabetes risk, and syndromic disorders. By capturing time-in-range, excursions, variability, and nocturnal patterns, CGM reveals pathophysiologic changes invisible to fasting glucose or HbA1c and provides actionable information for early intervention. Its ability to monitor glucose responses to routine meals, exercise, sleep, and illness makes it uniquely suited for both screening and ongoing monitoring in vulnerable pediatric populations. Integrating CGM alongside standard tests therefore supports earlier detection, more accurate risk stratification, and more timely preventive care.

Table 5 Cochrane-Oriented Quality Assessment (Only Applicable Studies)

Ref	Study Design	Risk-of-Bias Tool	Overall Assessment
6	Observational cohort (T2D CGM–HbA1c correlation)	ROBINS-I	Low–moderate risk
7	Observational cohort (TIR–HbA1c relationship)	ROBINS-I	Low–moderate risk
8	Prospective cohort, at-risk T1D	ROBINS-I	Low–moderate risk
9	Observational cohort, obesity and IR	ROBINS-I	Moderate risk
12	(Part of reference 33) RCT—DIAMOND Trial	RoB-2	Low risk
13	Interventional CGM risk-reduction study	RoB-2	Low–moderate risk
16	Observational CF cohort	ROBINS-I	Low–moderate risk
17	Postpartum dysglycemia CGM cohort	ROBINS-I	Moderate risk
18	Pregnancy CGM variability cohort	ROBINS-I	Moderate risk
19	Hemodialysis CGM cohort	ROBINS-I	Moderate risk
20	Pediatric obesity CGM cohort	ROBINS-I	Moderate risk
21	Adolescent obesity CGM/TRE cohort	ROBINS-I	Moderate risk

22	Prospective CF validation cohort	ROBINS-I	Low–moderate risk
24	β-thalassemia cohort (pediatric)	ROBINS-I	Moderate risk
25	β-thalassemia CGM–OGTT cohort	ROBINS-I	Moderate risk
26	Prospective at-risk T1D cohort	ROBINS-I	Low–moderate risk
27	Early at-risk pediatric cohort	ROBINS-I	Moderate risk
28	Prader–Willi syndrome metabolic cohort	ROBINS-I	Moderate risk
29	PWS transition cohort	ROBINS-I	Moderate risk
34	CF-related diabetes screening cohort	ROBINS-I	Low–moderate risk

The quality assessment indicates that most included studies are prospective or observational cohorts of low–moderate methodological rigor, with the main risks related to confounding and clinical heterogeneity rather than flaws in measurement or outcome assessment. These studies consistently demonstrate meaningful associations between CGM metrics and dysglycemia across diverse high-risk groups, providing a solid but non-causal evidence base. The few randomized or structured interventional trials included show low to low–moderate risk of bias, strengthening confidence in the physiological relevance and clinical utility of CGM-derived indicators such as TIR and glycemic variability. Overall, the evidence profile is sufficiently robust to support the review's conclusions while highlighting the need for more high-quality pediatric trials to definitively establish CGM's role in early detection and disease modification.

Overall, the compiled evidence demonstrates that CGM provides a richer and more sensitive assessment of glycemic abnormalities than traditional tests alone, particularly in high-risk pediatric groups where early dysglycemia is frequently missed by standard biochemical measurements.

4. Discussion

This review demonstrates that continuous glucose monitoring (CGM) provides a multidimensional characterization of glycemia that extends beyond the capabilities of fasting glucose, HbA1c, and oral glucose tolerance testing. The standardized CGM metrics outlined in international consensus statements— including time-in-range (TIR), time-above-range (TAR), time-below-range (TBR), mean glucose, and glycemic variability indices—form the basis for interpreting glucose patterns in clinical practice and research (1–5). These measures give insight into real-life glycemic dynamics, capturing fluctuations and physiological responses that cannot be inferred from single time-point laboratory values. This comprehensive perspective is particularly valuable in childhood and adolescence, when glucose regulation is influenced by growth, puberty, physical activity, and varied eating patterns.

An important finding across the literature is the strong but incomplete relationship between CGM-derived metrics and traditional biochemical measures. Mean glucose and TIR correlate with HbA1c in multiple populations (6,7), yet variation in TIR at the same HbA1c underscores inherent limitations of HbA1c as a sole marker of metabolic control. CGM metrics also align with OGTT-derived classifications of dysglycemia and with physiological indices of insulin sensitivity and β-cell function such as HOMA-IR, HOMA-β, and CGM-estimated disposition index (8,9,10, 18,34). These relationships demonstrate that CGM captures early metabolic dysfunction, particularly postprandial abnormalities and glycemic instability—before they manifest on routine biochemical tests, clarifying why CGM is a superior tool for early detection and physiological phenotyping.

The diagnostic advantage of CGM becomes especially evident in high-risk pediatric populations. In children with obesity, CGM often reveals reduced TIR, increased TAR, and frequent postprandial hyperglycemia despite normal fasting glucose or HbA1c (20,21). These abnormalities reflect early insulin resistance and impaired meal-related glucose handling that standard tests fail to detect. In cystic fibrosis, CGM identifies short-lived postprandial and nocturnal hyperglycemia years before CFRD is diagnosed via OGTT (16,22,23). These early excursions correlate with declining pulmonary function and nutritional status, making CGM a clinically meaningful tool for early therapeutic decisions. Among children with β-thalassemia major, CGM detects nocturnal hyperglycemia, peri-transfusion glucose surges, and variable dysglycemic patterns missed by OGTT and misrepresented by HbA1c due to altered erythrocyte lifespan (24,25). CGM thus identifies a greater proportion of youths with impaired glucose tolerance or diabetes compared with conventional methods.

Early type 1 diabetes represents another critical domain where CGM outperforms routine testing. Autoantibody-positive first-degree relatives frequently exhibit decreasing TIR, increasing TAR, and rising glycemic variability months or even years before OGTT criteria for dysglycemia are met (26,27). These early CGM abnormalities strongly predict progression to stage 3 type 1 diabetes and support the integration of CGM into screening, staging, and prevention trials. Similarly, in syndromic obesity and endocrine-metabolic disorders such as Prader–Willi syndrome, CGM reveals distinctive glucose disturbances related to hyperphagia, nocturnal eating, and altered satiety regulation even when fasting glucose and HbA1c remain normal (28,33). Collectively, these findings underscore that CGM provides actionable insight into disease-specific metabolic physiology and detects abnormalities that standard tests routinely overlook.

A recurring theme across all conditions is that CGM does not replace standard biochemical tests for the formal diagnosis of diabetes, which remain defined by regulatory criteria (30,31). However, CGM clearly emerges as the preferred method for screening, early detection, risk stratification, and treatment optimization in high-risk pediatric groups where dysglycemia is often intermittent, postprandial, or physiologically complex (6–9,16,20–22,24–27). HbA1c is limited in disorders affecting erythrocyte turnover (24,25), OGTT provides only a single-day snapshot (8,16,34), and fasting glucose overlooks meal-related and nocturnal abnormalities (20–23). CGM fills these gaps by capturing real-world glucose patterns influenced by daily life, illness, sleep, physical activity, and nutrition (1–3,14,15).

Looking ahead, several emerging applications highlight the expanding role of CGM in pediatric endocrinology. These include CGM-based prediction modeling for type 1 diabetes risk (8,26,27), estimation of insulin sensitivity and β-cell reserve from postprandial CGM data (10,14), and integration of CGM into hybrid closed-loop systems and digital phenotyping platforms (12,13,33). With ongoing improvements in device accuracy and accessibility (1,2,14,15), CGM is positioned to become central not only for diabetes management but also for metabolic screening in obesity, chronic systemic diseases, endocrine syndromes, and genetic disorders (20,21,24–29,32).

Overall, CGM provides a physiologically rich and clinically powerful assessment of glucose regulation that complements and enhances traditional testing (1–3,6,7). It identifies early dysglycemia across diverse pediatric disorders (16,20–29), clarifies metabolic heterogeneity (9,18,19), and supports timely, individualized care. Continued refinement of its applications will strengthen its role in early diagnosis, prevention, and precision endocrinology for children and adolescents (1,2,12,33).

5. Conclusion

Continuous glucose monitoring represents a major paradigm shift in the evaluation of glycemic health in children and adolescents, offering a dynamic and physiological understanding of glucose behavior that far exceeds the capabilities of traditional tests. By capturing real-life postprandial patterns, nocturnal excursions, and glycemic variability, CGM identifies early dysglycemia across diverse high-risk pediatric groups long before fasting glucose, HbA1c, or OGTT become abnormal. Its strong correlations with established metabolic indices, combined with superior sensitivity for early detection and disease staging, position CGM as an essential adjunct to conventional diagnostics and a powerful tool for individualized management. As CGM technology becomes more accessible, its integration into routine pediatric endocrine practice has the potential to enhance risk stratification, guide timely interventions, and ultimately improve long-term metabolic outcomes. Continued research should refine pediatric-specific thresholds and clarify how CGM-guided care can shape future screening and prevention strategies.

Recommendations

- Use CGM for early dysglycemia screening in high-risk pediatric groups where standard tests often miss early abnormalities.
- Combine CGM with traditional tests to improve diagnostic accuracy and guide individualized management.
- Develop pediatric-specific CGM targets and evaluate how CGM-guided care affects long-term metabolic outcomes.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no conflict of interest related to this work.

Statement of ethical approval

This article is a narrative review and did not involve human participants, identifiable patient data, or animal subjects; therefore, ethical approval and informed consent were not required.

Authors' Contributions

A.T.S. conceptualized the review, designed the outline, supervised data synthesis, and finalized the manuscript. S.A. and F.A. contributed to the literature search, evidence extraction, and drafting of the introduction and CGM metrics sections. A.E. and D.Y. reviewed comparative glycemic testing data and contributed to analysis of CGM–biochemical correlations. N.A. and N.H. assisted in compiling evidence for high-risk pediatric groups and contributed to critical revisions of the manuscript. N.S. supported background research and quality assessment of included studies. All authors reviewed and approved the final manuscript.

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