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Systematic review

Effectiveness of Continuous Glucose Monitoring for Glycemic Outcomes in Adults with Type 1 and Type 2 Diabetes

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Abstract
Continuous glucose monitoring (CGM) generates ambulatory glucose data that complement glycated haemoglobin (HbA1c) and capillary blood glucose monitoring by characterising glucose exposure, variability, and hypoglycemia. We systematically reviewed comparative studies of personal CGM in adults with type 1 or type 2 diabetes published from 2019 through October 2026. Randomised trials and comparative cohort studies were eligible when they evaluated real-time CGM (rtCGM), intermittently scanned CGM (isCGM), or flash glucose monitoring against self-monitoring of blood glucose, routine care, or another CGM modality and reported HbA1c, time in range (TIR), time below range (TBR), hypoglycemia, hyperglycemia, or glycemic variability. Pediatric-only, pregnancy-only, inpatient-only, accuracy-only, non-comparative studies, and automated insulin-delivery studies in which an independent CGM effect could not be isolated were excluded. Results were synthesised by diabetes type, insulin regimen, CGM strategy, comparator, and outcome because clinical and methodological diversity precluded a clinically meaningful pooled estimate. In type 1 diabetes, personal CGM compared with blood glucose monitoring reduced hypoglycemic exposure and modestly improved HbA1c in older adults. Among adults already using isCGM, switching to rtCGM improved TIR, HbA1c, and TBR over six months. In type 2 diabetes, personal CGM improved HbA1c and/or TIR among adults treated without insulin, with basal insulin, or with multiple daily injections; however, the magnitude and attribution of benefit depended on whether structured education or additional clinical support was part of the intervention. Findings for episodic and professional monitoring were less consistent and address different clinical strategies from sustained personal monitoring. Personal CGM is associated with clinically relevant improvements in glycemic outcomes in adults, particularly when the monitoring modality, insulin regimen, patient education, and follow-up support are aligned with the clinical objective.
Keywords. Continuous Glucose Monitoring, Type 1 Diabetes, Type 2 Diabetes, Glycated Haemoglobin.

Introduction

Glycated haemoglobin (HbA1c) is a central measure of long-term glycemic exposure, yet it cannot describe the timing, duration, or frequency of hyperglycemia and hypoglycemia. Two people with the same HbA1c may therefore have very different daily glucose profiles. Continuous glucose monitoring (CGM) measures interstitial glucose repeatedly and provides complementary metrics, including time in range (TIR), time below range (TBR), time above range, and glucose variability. International consensus recommendations identify a TIR of 70-180 mg/dL as a key glycemic metric for most adults, interpreted alongside HbA1c and clinically relevant time below and above range [1]. CGM can make glucose patterns visible at the time clinical decisions are made. Real-time CGM (rtCGM) provides current glucose values and can issue alerts, whereas intermittently scanned CGM (isCGM), also described as flash glucose monitoring, requires a scan to display current sensor information. Both approaches may improve self-management by supporting timely insulin adjustment, recognition of meal- and activity-related glucose excursions, and prevention of hypoglycemia. Their effectiveness, however, is not determined by the sensor alone. It may be modified by education, insulin regimen, baseline glycemic control, alert or scan use, access to follow-up, and the intensity of the comparator intervention. These distinctions are clinically important.

Adults with type 1 diabetes commonly require intensive insulin replacement and may prioritise prevention of hypoglycemia, whereas adults with type 2 diabetes have heterogeneous treatment regimens and may use CGM to guide lifestyle change, medication titration, or insulin use. A reduction in HbA1c does not answer the same clinical question as an increase in TIR or a reduction in TBR. Reviews of CGM should therefore preserve the outcome definition, population, monitoring strategy, and comparator rather than treating all sensor studies as interchangeable. The clinical question is therefore not simply whether CGM is useful. It is useful to ask which CGM method works best for a particular group of adults and compared with what alternative. Type 1 and type 2 diabetes differ in insulin needs and hypoglycemia risk. Within type 2 diabetes, people using no insulin, basal insulin, or multiple daily injections also have different treatment needs. A trial of rtCGM versus isCGM answers a different question from a trial of CGM versus routine finger-stick monitoring.

This review keeps these differences visible. It assesses type 1 and type 2 diabetes separately and groups studies by insulin regimen, CGM method, and comparator. Its main aim is to assess the effect of personal CGM on HbA1c and CGM-derived outcomes in adults. It also compares rtCGM with isCGM, describes hypoglycemia outcomes, and separates sustained personal CGM from episodic or professional monitoring.

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Methods

Review design and question

We conducted a systematic review with reference to PRISMA 2020. The review question used population, intervention, comparator, and outcome criteria. The population was adults aged 18 years or older with type 1 or type 2 diabetes. The interventions were personal rtCGM, isCGM, or flash glucose monitoring. The main comparators were SMBG/BGM, routine care, or another CGM method. The main outcomes were HbA1c, TIR, TBR, hypoglycemia, hyperglycemia, and glucose variability. We planned separate syntheses for type 1 and type 2 diabetes because insulin use, hypoglycemia risk, and treatment decisions differ between the two conditions.

Eligibility criteria

We included randomised controlled trials and comparative cohort studies of adults with type 1 or type 2 diabetes. Studies had to evaluate rtCGM, isCGM, or flash glucose monitoring and report at least one prespecified glycemic outcome. We accepted comparisons with SMBG/BGM, routine care, or another CGM method. We excluded pediatric-only, pregnancy-only, inpatient-only, accuracy-only, and non-comparative studies. We also excluded automated insulin-delivery studies when the independent effect of CGM could not be separated from the automated insulin intervention. Professional and episodic CGM studies were retained because they answer useful clinical questions, but were analysed separately from sustained personal CGM.

Information sources and search strategy

We searched PubMed from January 2019 through October 2026. The strategy combined controlled vocabulary and free-text terms for type 1 diabetes, type 2 diabetes, CGM, rtCGM, isCGM, flash glucose monitoring, SMBG/BGM, HbA1c, TIR, hypoglycemia, randomised trials, and comparative cohorts. We used the same main concepts when screening results from scholarly search tools and when checking citations of relevant reports. The search approach was designed to identify studies with an adult population, a defined CGM method, a meaningful comparison, and a reported glycemic outcome.

Study selection

We first screened titles and abstracts against the eligibility criteria and then assessed the full text of potentially relevant reports. Reports that described the same cohort or trial were linked in the evidence register so that participants were not counted more than once. We recorded selection decisions and the reason for exclusion at full-text assessment.

Data extraction

We used a structured evidence register to extract the study country, design, diabetes type, adult eligibility, insulin regimen, sample size, CGM method, comparator, follow-up, outcome definition, effect estimate, confidence interval when reported, analysis population, and reported funding or conflicts of interest. For each outcome, we recorded whether the result was a between-group comparison, a within-group change, or a descriptive result. This distinction was important because only a between-group estimate can directly answer the comparative review question.

Risk of bias assessment

For randomised studies, we assessed possible bias related to randomisation, deviations from the intended intervention, missing outcome data, outcome measurement, and selective reporting. We paid particular attention to open-label study designs, incomplete follow-up, differences in education or clinical contact between study groups, and whether the reported analysis matched the assigned intervention. These features were considered when interpreting each study rather than used to exclude evidence automatically.

Data synthesis

We used a narrative synthesis. A single pooled estimate would not be clinically useful because the studies differed in diabetes type, insulin regimen, CGM method, comparator intensity, education, follow-up, outcome definitions, and analysis populations. We therefore organised results by diabetes type and clinical stratum. We report adjusted between-group estimates and 95% confidence intervals when available. We do not treat a within-group change as proof of a comparative effect, and we do not compare TBR outcomes directly when the glucose thresholds differ.

Results

Overview of the evidence base

Most of the core evidence came from randomised trials, with selected comparative cohorts used for context. The studies enrolled adults with different diabetes duration, insulin regimens, baseline glucose control, and levels of hypoglycemia risk. We therefore analysed type 1 and type 2 diabetes separately. Within type 2 diabetes, we also separated non-insulin treatment, basal insulin, multiple daily injections, and professional or episodic monitoring. (Table 1) summarises the principal comparative studies, and (Figure 1) maps their reported outcome domains.

Table 1. Core comparative studies.

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Study	Population	Comparison	Follow-up	Key result	Role
Pratley 2020	T1D; age >=60; n=203	Personal CGM vs BGM	6 months	HbA1c −0.3; TBR <70 −1.9 points	Older-adult evidence
Visser 2021	T1D; prior isCGM; n=254	rtCGM vs isCGM	6 months	TIR +6.85; HbA1c −0.36; TBR <54 −0.35	Core modality RCT
Avari 2020 (I-HART)	T1D; high hypoglycemia risk; n=40	rtCGM vs isCGM	8 weeks	Fewer hypoglycemia episodes and favourable hypoglycemia-related variability measures with rtCGM	High-risk T1D RCT
Martens 2021	T2D; basal insulin; n=175	rtCGM vs BGM	8 months	HbA1c −0.4; TIR +15 points	Core basal-insulin RCT
Wada 2020	T2D; no insulin; n=100	isCGM vs SMBG	24 weeks	HbA1c −0.29 after 12-week exposure	Core non-insulin RCT
Aronson 2023	T2D; no insulin; n=115	isCGM plus DSME vs DSME	16 weeks	TIR +9.9; HbA1c −0.3 points	Balanced-education RCT
Kim 2024	T2D; MDI; n=159	isCGM plus education vs BGM	24 weeks	Package HbA1c −1.00 vs −0.58%	Sensor-plus-education
Furler 2020	T2D; mixed therapy; n=299	Professional flash vs usual care	12 months	HbA1c not significant; TIR +7.9	Separate professional strategy

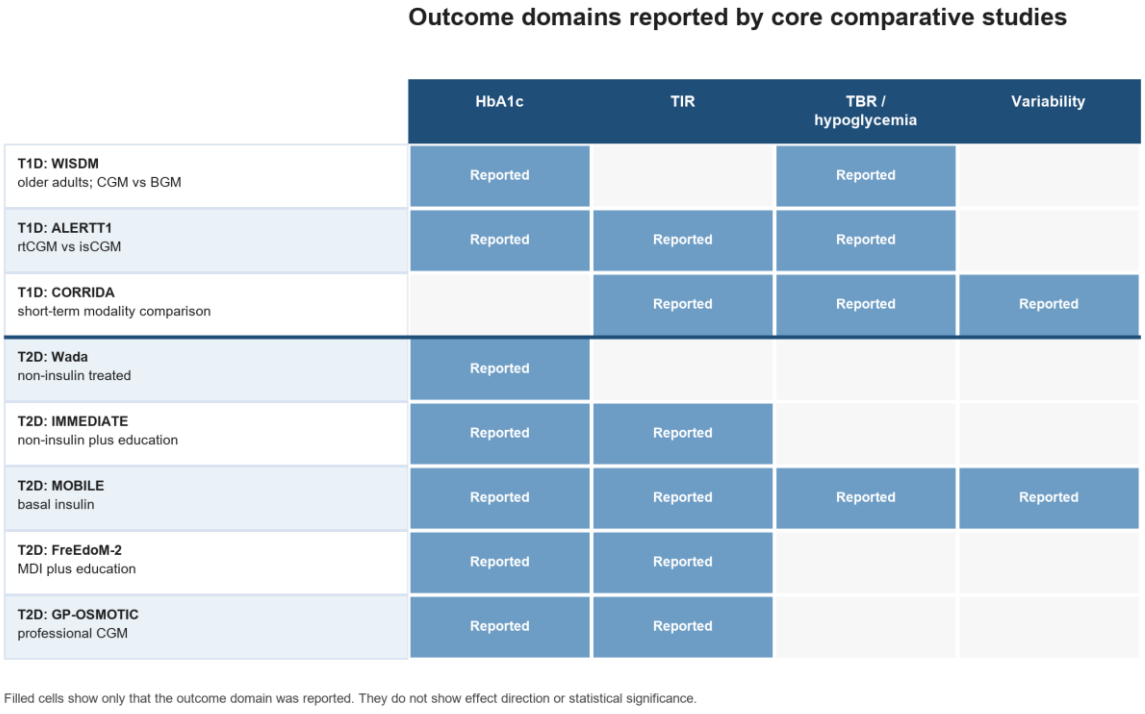


Figure 1. Outcome domains reported by core studies.

Type 1 diabetes

The WISDM randomised trial compared CGM with standard BGM in 203 adults aged 60 years or older with type 1 diabetes. At six months, HbA1c was 0.3 percentage points lower with CGM (95% CI −0.4 to −0.1), and TBR below 70 mg/dL was 1.9 percentage points lower (95% CI −2.8 to −1.1).[2] In ALERTT1, 254 adults who already used isCGM were assigned to rtCGM or continued isCGM. The rtCGM group increased TIR by 6.85 percentage points (95% CI 4.36 to 9.34), reduced HbA1c by 0.36 percentage points, and spent less time below 54 mg/dL at six months.[3] CORRIDA also found higher TIR and lower TBR with rtCGM during short exercise and home phases [4]. I-HART showed benefit for several hypoglycemia measures in adults at high risk of hypoglycemia, although some hyperglycemia and variability measures favoured isCGM.[5] A small four-week study soon after type 1 diabetes diagnosis found no significant difference in TIR or TBR and was too short to assess HbA1c [6].

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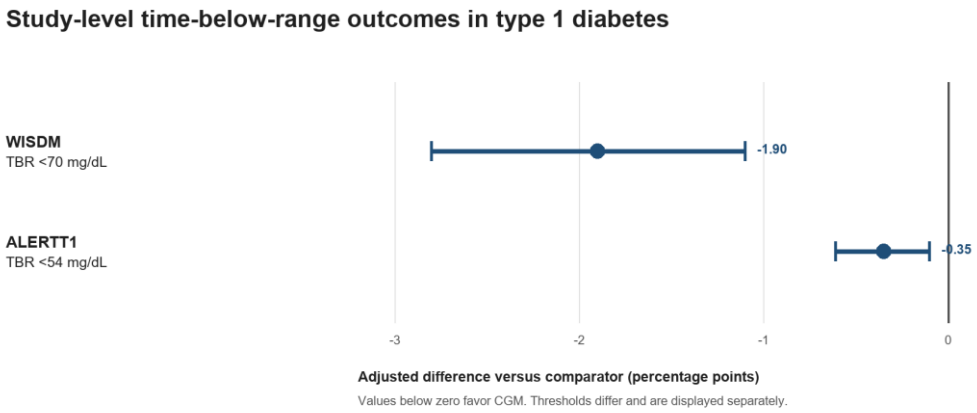


Figure 2. Hypoglycemia outcomes in type 1 diabetes.

Type 2 diabetes

In adults with type 2 diabetes who did not use insulin, Wada and colleagues found an adjusted HbA1c difference of −0.29 percentage points at 24 weeks after 12 weeks of flash monitoring compared with SMBG (95% CI −0.54 to −0.05) [7]. In the IMMEDIATE trial, isCGM plus diabetes self-management education improved TIR by 9.9 percentage points (95% CI 2.5 to 17.3) and produced a 0.3 percentage-point greater HbA1c reduction than the same education alone.[8] In adults using basal insulin without prandial insulin, MOBILE found that rtCGM lowered HbA1c by 0.4 percentage points (95% CI −0.8 to −0.1) and increased TIR by 15 percentage points (95% CI 8 to 23) at eight months compared with BGM [9]. In FreEdoM-2, the significant HbA1c contrast represented CGM plus repeated structured education versus BGM plus conventional education. The two conventionally educated groups had similar descriptive changes, so this result should be read as the effect of a care package rather than the effect of the sensor alone [10].

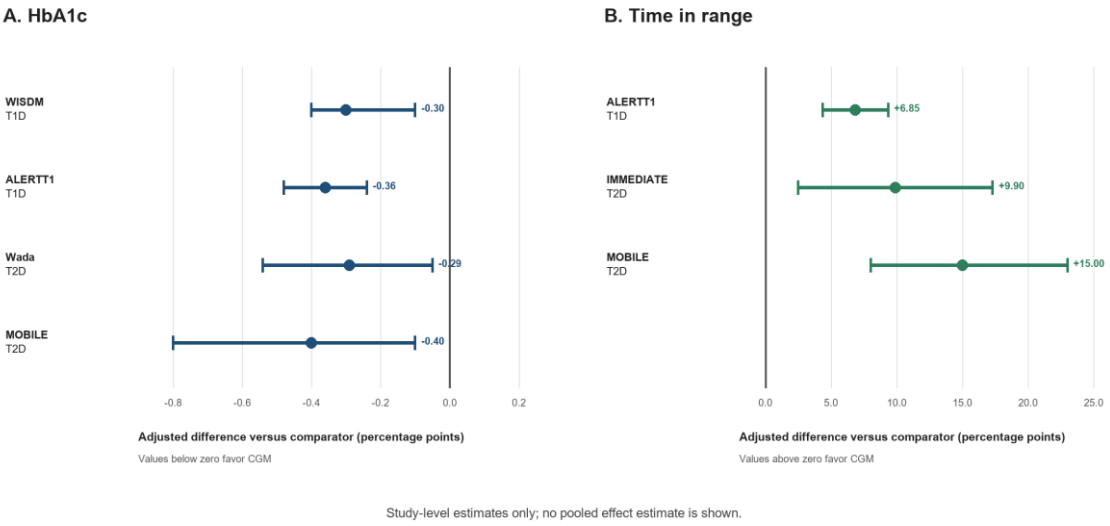


Figure 3. Selected HbA1c and time-in-range effects.

(Figure 4) Summarises the direction of the reported comparative findings without converting study-specific estimates into a pooled effect.

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Direction of comparative glycemic findings by clinical stratum

	HbA1c	TIR	TBR / hypoglycemia	Interpretation
T1D: WISDM	Improved	NR	Improved	CGM vs BGM; older adults
T1D: ALERTT1	Improved	Improved	Improved	rtCGM vs isCGM
T1D: CORRIDA	NR	Improved	Improved	Short-term modality comparison
T2D: Wada	Improved	NR	NR	Flash CGM vs SMBG; no insulin
T2D: IMMEDIATE	Improved	Improved	NR	Same education in both groups
T2D: MOBILE	Improved	Improved	Improved	rtCGM vs BGM; basal insulin
T2D: FreEdoM-2	Improved*	Improved*	NR	Sensor plus structured education
T2D: GP-OSMOTIC	Mixed	Improved	NR	Professional monitoring

Improved = statistically favorable comparative finding; Mixed = outcome or follow-up dependent; NR = not reported. *Interpret as a sensor-plus-education package.

Figure 4. Direction of comparative glycemic findings.

Episodic and professional monitoring

Episodic and professional CGM are different care strategies from sustained personal CGM. In the COMMITED pilot, three 10-day rtCGM sessions over 12 weeks did not produce a significant HbA1c difference versus SMBG, and the apparent benefit was not sustained at month 9 [11]. In GP-OSMOTIC, professional flash monitoring every three months improved HbA1c at six months but not the primary HbA1c outcome at 12 months; TIR improved at 12 months.[12] LIBERATES enrolled adults after myocardial infarction, so it is better viewed as evidence for a specific clinical setting rather than routine outpatient type 2 diabetes care [13].

Discussion

Overall, CGM improved important glycemic outcomes in adults, but the size and type of benefit differed between groups. In type 1 diabetes, the clearest findings were more time in the target range and less time in hypoglycemia. This was most evident when rtCGM with alerts replaced isCGM in established users and when CGM replaced BGM in older adults. The two studies shown in Figure 3 used different TBR thresholds. Their findings both favour CGM, but they should not be combined as though they measured the same outcome. In type 2 diabetes, personal CGM improved HbA1c and/or TIR in studies of people using no insulin, basal insulin, and multiple daily injections. The findings are especially useful when read with the study design in mind. A comparison with routine BGM tests the value of introducing CGM. A comparison with the same education in both groups tests the added value of the sensor. A comparison that gives more education or clinical contact to the CGM group instead tests a broader care package. The FreEdoM-2 and IMMEDIATE trials illustrate this difference.

Education and clinical contact were important parts of several interventions. In FreEdoM-2, isCGM was introduced with repeated structured education, so the result should not be described as a sensor-only effect. In IMMEDIATE, both groups received the same education, which gives a clearer estimate of the added value of isCGM. These distinctions are important for clinical implementation, reimbursement, training, and service planning. The studies were not alike enough to support one pooled estimate that would be clinically meaningful for all adults. They differed in diabetes type, insulin regimen, sensor generation, alert use, education, comparator intensity, follow-up, outcome definitions, and analysis population. These differences do not negate the observed benefits; rather, they determine the clinical question each study can answer. Table 2summarises the interpretation boundaries used in this review. (Figure 5) shows the clinical framework used to interpret studies by diabetes type, monitoring strategy, comparator, and outcome.

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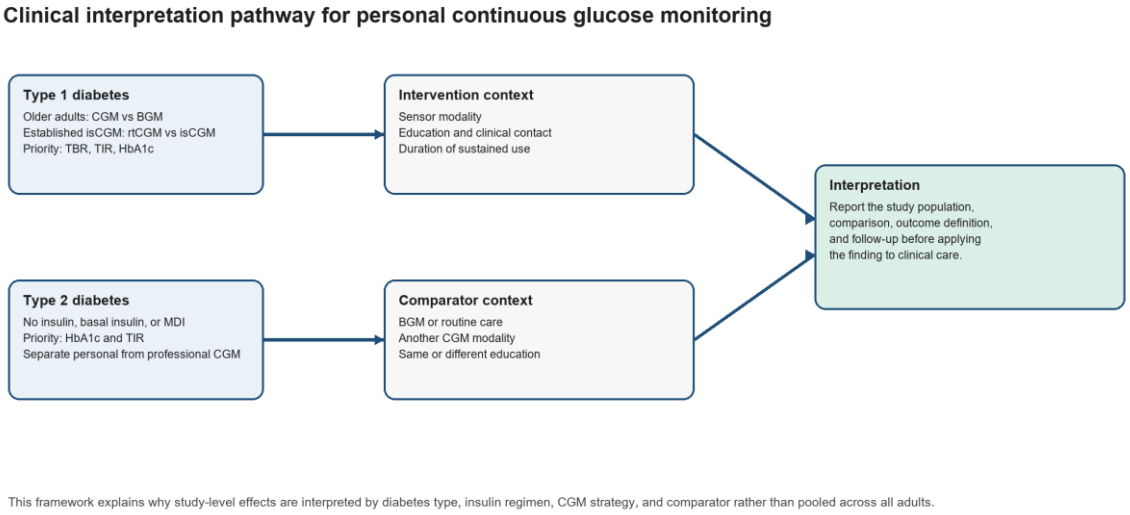


Figure 5. Clinical interpretation pathway for CGM evidence.

Table 2. Interpretation framework.

Clinical stratum	Supported interpretation	Do not infer
Type 1 older adults	CGM reduced hypoglycemic exposure and modestly improved HbA1c versus BGM.	A universal effect in all ages.
Type 1 established isCGM users	rtCGM improved TIR and hypoglycemia-related outcomes.	Effect of starting CGM versus BGM.
Type 2 no insulin	Personal CGM/isCGM improved HbA1c and/or TIR in selected adults.	Durable effect from short episodic use.
Type 2 basal insulin	Sustained rtCGM improved HbA1c and TIR versus BGM.	Equivalence to professional monitoring.
Professional or episodic monitoring	Supports contextual clinician-supported monitoring.	Effect of continuous personal CGM.

Conclusion

Personal CGM is an effective component of adult diabetes care when it is matched to the patient's clinical objective and supported by appropriate education and follow-up. In type 1 diabetes, the most consistent benefits are reductions in hypoglycemic exposure and improvement in TIR; rtCGM provides additional benefit for adults already using isCGM in comparative evidence. In type 2 diabetes, sustained personal CGM improves HbA1c and/or TIR across clinically distinct groups, including adults treated without insulin, with basal insulin, and with multiple daily injections. These findings support individualised implementation rather than a single CGM strategy for all adults. Decisions about access and use should account for diabetes type, insulin regimen, the selected monitoring method, ability to act on glucose data, and the glycemic outcome most relevant to the individual patient.

Declarations

Author contributions

Rima Ahmed Salem Elganduz contributed to study selection, data extraction, interpretation of findings, and manuscript drafting. Nada Khaled Elbibi contributed to study conception and critical scientific revision of the manuscript. Both authors approved the final version and agree to be accountable for all aspects of the work.

Acknowledgments

Not applicable.

Conflict of interest

Rima Ahmed Salem Elganduz and Nada Khaled Elbibi declare that they have no competing interests.

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Ethics approval and consent to participate

Not applicable. This systematic review used published data only.

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Data availability

All data synthesized in this review were derived from published reports. The structured evidence register and extracted outcome data should be retained by the authors and made available on reasonable request, subject to source-article copyright restrictions.

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