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REVIEW ARTICLE

Continuous glucose monitoring and glycaemic variability in acute ischaemic stroke: a systematic review with narrative synthesis

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Abstract

Dysglycaemia is common after acute ischaemic stroke and is associated with adverse outcomes. Continuous glucose monitoring (CGM) provides frequent interstitial glucose measurements and may identify glycaemic variability and excursions missed by intermittent testing. This systematic review evaluated CGM feasibility, accuracy, glycaemic patterns and clinical associations in acute ischaemic stroke. MEDLINE, Embase and CENTRAL were searched to January 2026 for studies of adults undergoing CGM within seven days of stroke onset or admission. Risk of bias was assessed using RoB 2 or the Newcastle–Ottawa Scale, and certainty of evidence using GRADE. Seven studies enrolled 595 participants; monitoring lasted 24 h to 4.5 days. Mean glucose ranged from approximately 118 to 149 mg/dL and coefficient of variation from 4% to 21%. Observational studies provided low- or very-low-certainty evidence that greater hyperglycaemic exposure or variability was associated with death or dependency, unfavourable outcome after thrombectomy and reduced in-hospital neurological improvement. An unadjusted time-above-range difference between functional-trajectory groups was not independently significant after adjustment. One diabetes-only randomised trial evaluated a multi-component intervention combining structured nursing, insulin-pump therapy and CGM. Two-week neurological, motor, self-care and quality-of-life measures favoured the intervention, but modified Rankin Scale and longer-term outcomes were not reported, and the effect of CGM could not be isolated. CGM appears feasible, but evidence is heterogeneous and insufficient to support routine implementation. Standardised metrics and adequately powered multicentre trials are required.

Keywords Acute ischaemic stroke · Continuous Glucose Monitoring · Glycaemic variability · Time in range · Dysglycaemia · Glycaemic control

Introduction

Acute ischaemic stroke remains a major cause of death and disability despite advances in reperfusion therapy [1, 2]. Although reperfusion improves outcome in selected patients, many remain dependent, and identifying modifiable physiological processes that influence infarct evolution and recovery remains important.

Dysglycaemia occurs in people with and without diabetes after stroke and reflects stress responses, inflammation, altered nutrition and pre-existing metabolic disease [3, 4]. Admission and persistent hyperglycaemia are associated with mortality, infarct expansion and poor functional outcome [3–6], and may increase haemorrhagic transformation risk after reperfusion [7]. However, glucose physiology is dynamic: intermittent capillary testing may miss short-lived hyperglycaemia, clinically silent hypoglycaemia and rapid fluctuations that could be relevant to tissue injury or recovery.

Glycaemic variability (GV) describes glucose fluctuations over time and has been associated with adverse outcomes in critical illness [8, 9]. Continuous glucose monitoring (CGM) provides frequent interstitial glucose measurements and permits calculation of mean glucose, standard deviation, coefficient of variation, fluctuation indices and time-in-range measures [10–12]. It may

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therefore characterise glycaemic exposure more fully than isolated bedside measurements.

In acute stroke, CGM could have two complementary roles: identifying high-risk glycaemic phenotypes and supporting interventions designed to reduce harmful excursions without causing hypoglycaemia. Evidence remains fragmented across small cohorts using different devices, monitoring periods, metrics and outcomes. We therefore reviewed CGM feasibility and accuracy in acute ischaemic stroke, described early glucose patterns and synthesised associations with neurological and functional outcomes, including interventional evidence.

Methods

Protocol and registration

This review followed PRISMA 2020 guidance [13]. The protocol was prospectively registered with PROSPERO (CRD42025113766).

Eligibility criteria

We included randomised and non-randomised intervention studies, cohort studies and case-control studies involving adults (≥18 years) with neuroimaging-confirmed acute ischaemic stroke and CGM initiated within seven days of onset or admission. Any commercially available CGM platform and monitoring duration were eligible. Observational studies did not require a comparator; intervention studies required standard care or another monitoring strategy. Primary outcomes were functional outcome at approximately three months, mortality and neurological severity. Secondary outcomes included completion, accuracy, safety, GV indices and time-in-range measures. We excluded reports published before 2010, non-English publications, case reports or series with fewer than 10 participants, editorials and conference abstracts without full text.

Protocol amendment

During full-text screening, eligibility criteria were amended before data extraction and synthesis. Mixed ischaemic and haemorrhagic stroke cohorts were retained when ischaemic stroke comprised more than half of participants and outcomes could not be disaggregated (Wada 2018; Theodorou/Palaiodimou 2021). These are identified throughout as mixed cohorts. The sole randomised trial (Wu 2025) was retained despite unclear CGM initiation timing because it provided the only interventional evidence. Excluding the mixed cohorts substantially reduced adjusted prognostic evidence, although findings from ischaemic-stroke-only cohorts were directionally consistent (Supplementary S4 File).

Information sources and search strategy

MEDLINE, Embase and CENTRAL were searched from 01 January 2010 to 31 January 2025 and updated on 28 January 2026. Trial registries, relevant stroke conference proceedings, preprint servers and reference lists of included studies were also searched. Controlled vocabulary and free-text terms combined acute stroke, CGM and glycaemic variability concepts. The complete database strategies, limits and update results are provided in Supplementary S1.

Study selection

Two reviewers (JV, RPJ) independently screened titles and abstracts and subsequently assessed full texts. Disagreements were resolved by discussion or, when necessary, third-reviewer arbitration (KC). Reasons for full-text exclusion were recorded. No formal calibration exercise was performed before screening commenced.

Data extraction

Two reviewers independently extracted study design, setting, participant characteristics, diabetes status, baseline NIHSS, reperfusion therapy, CGM device and protocol, glucose metrics and clinical outcomes using a standardised Excel form. Adjusted estimates were prioritised where available. Disagreements were resolved by consensus or KC adjudication. Means and standard deviations were estimated from medians and dispersion measures when required [14], and graphical data were digitised where applicable. Derived values were identified as such and were not combined with directly reported effect estimates.

Risk of bias and certainty assessment

Randomised trials were assessed using RoB 2 [15], and observational studies using the Newcastle–Ottawa Scale [16]. Two reviewers completed assessments independently and resolved disagreements by consensus. Certainty of evidence for each principal outcome was evaluated using GRADE [17], considering risk of bias, inconsistency, indirectness, imprecision and publication bias. Observational evidence started at low certainty.

Data synthesis

Formal meta-analysis and I^2 estimation were not undertaken because studies differed substantially in populations, CGM devices, monitoring duration, GV definitions, glucose thresholds, outcomes and analytical methods. No common effect measure was available from at least two sufficiently comparable studies addressing the same PICO question [18]. Pooling would therefore have produced a clinically difficult-to-interpret summary and potentially misleading precision.

Findings were synthesised narratively, with adjusted estimates prioritised over unadjusted comparisons. Comparable descriptive glucose metrics are presented as study-level ranges rather than pooled means. Prognostic associations, feasibility or accuracy findings and interventional effects were considered separately.

Results

Study selection and characteristics

Seven studies enrolled 595 participants and met the inclusion criteria. Included designs comprised one RCT, five prospective cohorts and one pilot feasibility study (Table 1). Publication years ranged from 2018 to 2025, with studies conducted in the UK, China, Greece, Japan and the Netherlands. Figure 1 presents the original search flow and the January 2026 search update.

Participant characteristics

Participants were aged approximately 54–76 years. Diabetes prevalence ranged from 21% to 100%; the randomised trial included only patients with diabetes. Most general stroke cohorts had mild-to-moderate deficits, whereas thrombectomy cohorts were more severe. Three studies involved thrombectomy-treated patients, including one outcome-focused cohort and two feasibility or accuracy studies. This clinical heterogeneity limits direct comparison and generalisability.

CGM platforms and monitoring protocols

Dexcom G6, Medtronic iPro2 and FreeStyle Libre systems were used, alongside pump-integrated CGM in the randomised trial. Sensors were placed on the abdomen or upper arm. Monitoring lasted 24 h to approximately 4.5 days and generally began within 24–48 h of admission or stroke onset. Some devices were factory calibrated, whereas retrospective Medtronic systems required capillary calibration.

Table 1 Characteristics of included studies

Study	Design	Country	*N*	Age, years	Diabetes	CGM device	Duration	Population	Outcomes
Preechausuk 2025 [18]	Prospective cohort	UK	67	72 ± 14	28%	Dexcom G6	~4.5 days	Mixed AIS	Functional trajectory; GV metrics
Wu 2025 [19]	RCT	China	100	54 ± 10	100%	Pump-integrated CGM	Not stated	Diabetes only (AIS); protocol amendment†	NIHSS, FMA, ESCA, SF-36 at 2 weeks
Theodorou (Palaiodimou) 2021 [20]	Prospective cohort	Greece	62	65 ± 10	Not fully reported	Medtronic iPro2	70 h	Mixed AIS 77% + ICH 23%; protocol amendment‡	In-hospital neurological improvement
Wada 2018 [21]	Prospective cohort	Japan	100	70 ± 13	26%	Medtronic iPro2	72 h	Mixed AIS 58% + ICH 42%; protocol amendment‡	Death/dependency at 3 months
Shi 2024 [22]	Prospective cohort	China	141	71 (60–77)	32%	Medtronic iPro2	4.5 days	AIS + EVT	Feasibility, accuracy
Kersten 2024 [23]	Prospective cohort	Netherlands	102	76 (61–83)	30%	FreeStyle Libre 2	> 72 h	LVO + EVT	90-day outcome
Kersten 2023 [24]	Pilot feasibility	Netherlands	23	74 ± 16	30%	FreeStyle Libre	24 h	Mixed AIS	Feasibility, accuracy

†Wu 2025: CGM initiation timing relative to stroke onset was unclear; the study was retained as the sole interventional evidence following protocol amendment

‡Theodorou/Palaiodimou 2021 enrolled 48 ischaemic and 14 haemorrhagic strokes; Wada 2018 enrolled 58 ischaemic and 42 haemorrhagic strokes. Both mixed cohorts were retained following protocol amendment. A qualitative applicability assessment is presented in Supplementary S4 File

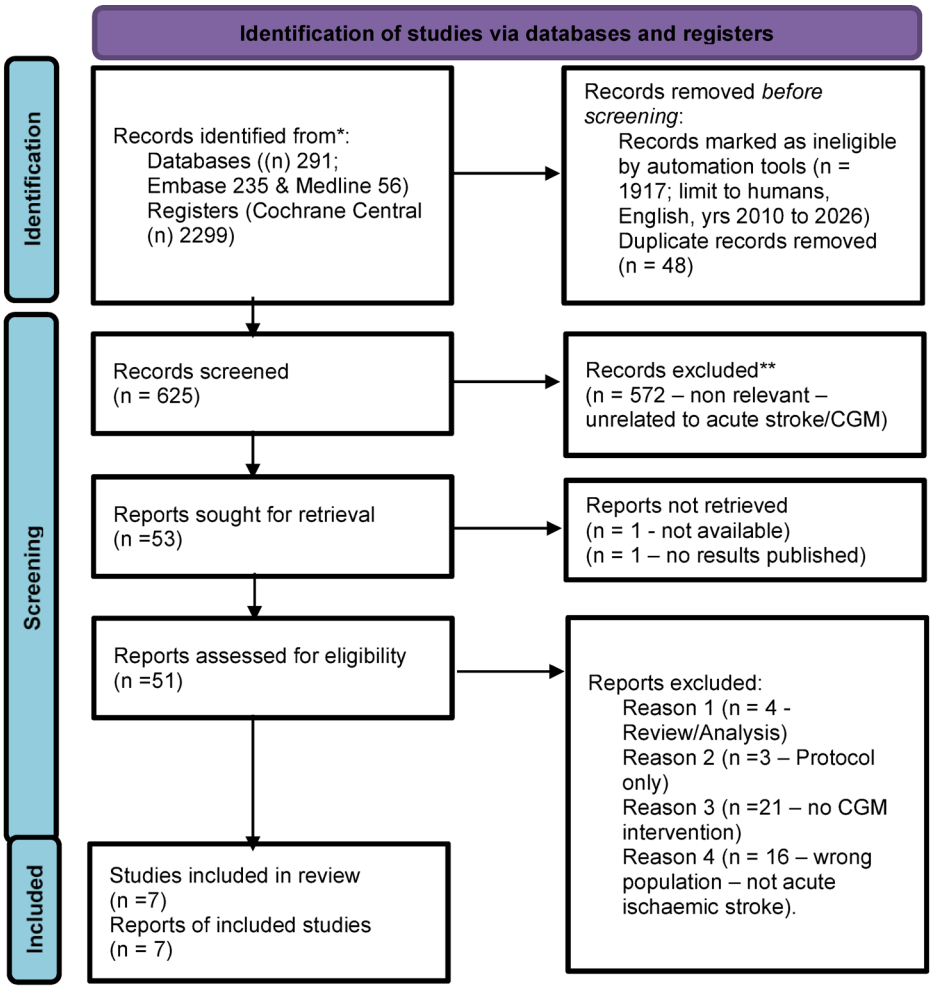

Fig. 1 PRISMA 2020 flow diagram of study selection

The main flow reports the original search. The January 2026 update identified 50 additional records but no additional eligible studies.

Feasibility, safety, and accuracy

Reported completion rates were approximately 85–90%. A thrombectomy pilot completed the planned 24-hour monitoring period in 87% of participants [19]. No serious device-related adverse events or documented disruption to thrombolysis, thrombectomy or routine stroke-unit care were reported. However, feasibility definitions varied and most studies did not systematically quantify sensor loss, skin reactions, nursing workload or reasons for incomplete recording.

Accuracy data were limited. Kersten 2023 found all paired sensor and reference readings within clinically acceptable Parkes error-grid zones A/B [19]. Shi 2024 reported a mean absolute relative difference of approximately 14.6%, with more than 98.9% of paired values in clinically acceptable error-grid zones [20]. Accuracy at the extremes of the glycaemic range, during altered perfusion and in critically ill patients remains insufficiently characterised.

Hypoglycaemia reporting was inconsistent but suggested that intermittent testing may miss clinically relevant events. Wada et al. reported at least one glucose value < 4 mmol/L in 20% of participants [21]. Theodorou/Palaiodimou identified 32 CGM-detected hypoglycaemic episodes in 17 non-diabetic participants, none detected by concurrent finger-prick testing [22]. Shi et al. detected level-1 hypoglycaemia in 43.3% by CGM versus 7.8% by point-of-care testing [20], while Kersten 2023 reported 13% time below range [19]. Thresholds and denominators differed, so these results cannot be pooled, but they support standardised prospective reporting of time below 3.9 and 3.0 mmol/L, symptomatic events and treatment-related hypoglycaemia.

Glucose dynamics and variability

Across studies reporting numerical values, mean glucose ranged from approximately 118–149 mg/dL and CV from 4% to 21% (Table 2). These ranges are descriptive rather

Table 2 Glucose dynamics, variability metrics, and primary GV metric reported across included studies

Study	*N*	Mean glucose, mg/dL	CV, %	TIR, %	TAR, %	Primary metric(s)	Notes
Preechausuk 2025 [18]	67	121–140*	16.4–18.3*	85.7–96.7*	0.0–11.1*	CV, SD, TAR	*Trajectory subgroups reported
Theodorou 2021 [20]	62	118	NR	NR	NR	MAG	MAG used for outcome models
Wada 2018 [21]	100	149	15.5	NR	NR	AUC > 8 mmol/L; Time-ratio > 8 mmol/L; Mean glucose	Hyperglycaemia exposure indices
Shi 2024 [22]	141	130	21.25	NR	NR	CV, Mean glucose	Feasibility/accuracy focus
Kersten 2024 [23]	102	126	4.0	NR	NR	Time-ratio > 7.8 mmol/L; Mean glucose	EVT cohort
Kersten 2023 [24]	23	NR	NR	NR	13%	CV, SD, TBR	24-hour monitoring only

NR = not reported. CV = coefficient of variation; TIR = time in range (3.9–10 mmol/L); TAR = time above range (> 10 mmol/L); TBR = time below range (< 3.9 mmol/L); MAG = mean absolute glucose; AUC = area under curve above threshold; SD = standard deviation; *Values represent the range across the functional-trajectory subgroups; they are not overall cohort estimates. The asterisk does not denote statistical significance

than pooled estimates. Direct comparison was limited by differences in monitoring duration, patient selection, device type and glucose thresholds. Several studies reported subgroup values or alternative indices such as MAG, AUC above threshold and study-specific time-above-threshold measures.

Time-in-range measures were incompletely and inconsistently reported. In Preechasuk et al., participants who deteriorated from independent to dependent status had higher mean glucose, SD and time above range than those who remained independent; however, only baseline NIHSS remained independently associated with functional trajectory [23].

Associations with outcomes

Adjusted prognostic associations are summarised in Table 3. Outcomes and exposure metrics differed sufficiently that each finding should be interpreted as evidence from an individual cohort rather than as a common pooled effect.

Haemorrhagic transformation was not reported as a formal post-CGM outcome in any included study, despite its clinical relevance to hyperglycaemia and reperfusion therapy. Cerebral oedema and infarct growth were also rarely evaluated, leaving important mechanistic endpoints underrepresented.

In Wada 2018, high mean glucose, area under the curve above 8 mmol/L and time above 8 mmol/L independently predicted death or dependency at three months, with adjusted ORs ranging from 3.03 to 4.67 [21]. The cohort included both ischaemic and haemorrhagic stroke, so indirectness limits application to a purely ischaemic population.

Kersten 2024 found that admission hyperglycaemia > 10 mmol/L predicted unfavourable 90-day outcome after thrombectomy (adjusted OR 2.7, 95% CI 1.1–6.7) [24]. CGM-derived time-above-threshold measures were explored, but admission glucose rather than a validated GV metric provided the principal adjusted association.

In the mixed Theodorou/Palaiodimou cohort, higher mean absolute glucose was associated with reduced odds of in-hospital neurological improvement (adjusted OR 0.135, 95% CI 0.024–0.751) [22]. The study reported short-term neurological change rather than three-month disability, and inclusion of haemorrhagic stroke introduces indirectness.

Preechasuk et al. reported higher unadjusted mean glucose, SD and time above range in participants who became dependent [23]. Nevertheless, GV measures were not independently associated with trajectory after adjustment, emphasising the dominant influence of baseline stroke severity.

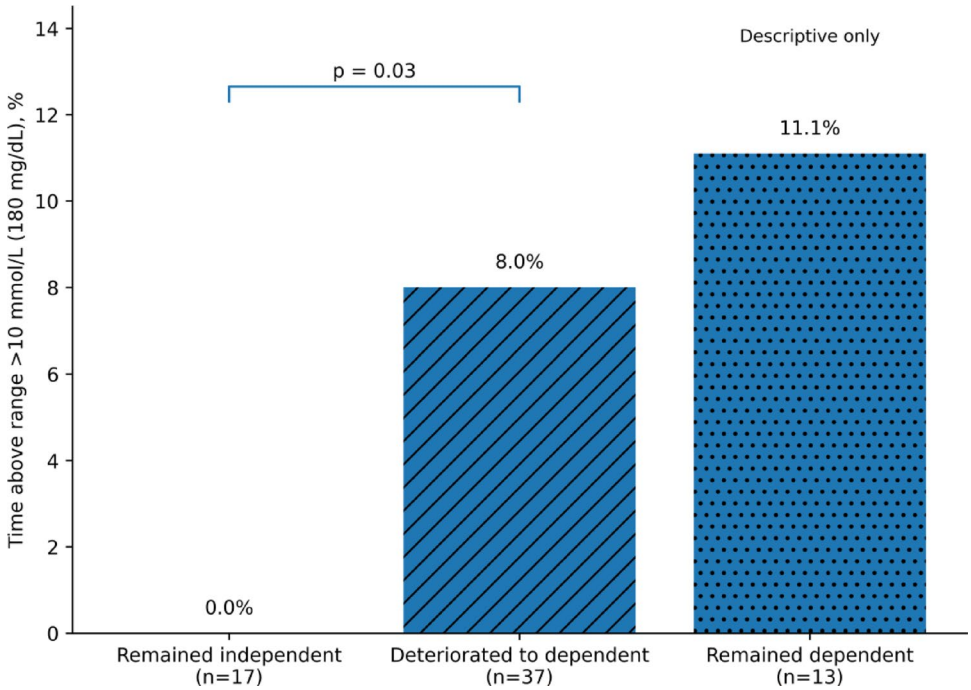

Fig. 2 Time above range > 10 mmol/L (180 mg/dL) according to functional-status trajectory. TAR was higher in participants who deteriorated from independent to dependent status than in those who remained independent in an unadjusted comparison ($p = 0.03$). The remained-dependent group is shown descriptively; no pairwise comparison involving this group was reported. Data from Preechasuk et al. [23]

Table 3 Summary of key associations between CGM metrics and clinical outcomes

Outcome	Study	Metric	Adjusted effect (95% CI)	Interpretation
Death/dependency at 3 months (mRS 3–6)	Wada 2018 [21]	High mean glucose	OR 3.03 (1.26–7.27)	Higher exposure associated with worse outcome
Death/dependency at 3 months (mRS 3–6)	Wada 2018 [21]	High AUC > 8 mmol/L	OR 3.82 (1.53–9.53)	Hyperglycaemia burden associated with worse outcome
Death/dependency at 3 months (mRS 3–6)	Wada 2018 [21]	High time-ratio > 8 mmol/L	OR 4.67 (1.78–12.27)	Time above threshold associated with worse outcome
Unfavourable outcome at 90 days (mRS 3–6)	Kersten 2024 [23]	Admission hyperglycaemia (> 10 mmol/L)	OR 2.7 (1.1–6.7)	Hyperglycaemia associated with poor outcome after EVT
In-hospital neurological improvement	Theodorou 2021 [20]	Higher MAG	Adj OR 0.135 (0.024–0.751)	Higher variability independently associated with reduced in-hospital improvement; in-hospital outcomes only — no 3-month data reported

OR = odds ratio; CI = confidence interval; AUC = area under curve above 8 mmol/L; MAG = mean absolute glucose; TAR = time above range (> 10 mmol/L); TBR = time below range (< 3.9 mmol/L); mRS = modified Rankin Scale; NIHSS = National Institutes of Health Stroke Scale

Interventional evidence

One diabetes-only randomised trial (n = 100) compared enhanced nursing plus CGM-guided insulin-pump management with standard care [25]. At two weeks, the intervention group had statistically better NIHSS, motor function, self-care and quality-of-life scores and lower fasting glucose (Table 4). mRS, mortality and longer-term outcomes were not reported. Because the intervention combined nursing, CGM and insulin-pump therapy, the independent contribution of CGM cannot be determined. The single-centre design, unclear allocation concealment, short follow-up and uncertain timing of CGM initiation further limit generalisability.

Risk of bias and certainty of evidence

The randomised trial was judged to have some concerns under RoB 2 because allocation concealment was unclear and participant or staff blinding was not feasible. Observational studies were generally rated low or moderate risk on the Newcastle–Ottawa Scale, but residual confounding, selective reporting and small samples remained important. GRADE certainty was low or very low for all principal outcomes because most were supported by single studies, with downgrading for imprecision, indirectness or risk of bias (Supplementary S3 and Table S6).

Table 4 Interventional evidence (RCT): Wu 2025 [25]

Outcome	Assessment timepoint	Intervention (median [IQR])	Control (median [IQR])	Test statistic	*p*-value
NIHSS	2 weeks	6.00 [4–7]	7.00 [5.75–8.25]	Z = − 4.176	< 0.001
FMA (motor function)	2 weeks	67.00 [60.75–72]	58.50 [52.5–64.25]	Z = − 4.782	< 0.001
ESCA (self-care capacity)	2 weeks	Not reported numerically	—	Z = − 5.998	< 0.001
SF-36 (all domains)	2 weeks	Not reported numerically	—	—	All < 0.001
Fasting glucose (mmol/L)	2 weeks	—	—	MD − 0.87 (95% CI − 1.24 to − 0.50)	< 0.001

mRS was not reported in this study. All outcomes were assessed at 2 weeks only; no data were available for longer follow-up. The intervention comprised whole-course fine nursing plus CGM-guided insulin-pump management, with fasting glucose targeted at 4.4–7.0 mmol/L and 2-hour postprandial glucose below 10.0 mmol/L; benefit cannot be attributed to CGM monitoring alone. NIHSS = National Institutes of Health Stroke Scale; FMA = Fugl-Meyer Assessment; ESCA = Exercise of Self-Care Agency Scale; SF-36 = Short Form Health Survey; MD = mean difference; IQR = interquartile range; mRS = modified Rankin Scale

Discussion

Principal findings

This review identified a small and heterogeneous evidence base comprising observational prognostic studies, descriptive or feasibility studies, and one diabetes-only randomised trial. Observational cohorts associated higher GV or greater hyperglycaemic exposure with adverse neurological or functional outcomes. Preechasuk et al. reported an unadjusted TAR difference between functional-trajectory groups, but glycaemic metrics were not independently associated with trajectory after adjustment. These signals should be regarded as hypothesis-generating rather than evidence that modifying GV improves outcome.

The randomised trial reported favourable two-week outcomes after a multi-component intervention combining enhanced nursing, CGM and insulin-pump therapy. CGM's independent contribution cannot be isolated, and neither mRS nor longer-term outcomes were assessed. Overall certainty was low or very low because most outcomes relied on single, small studies and residual confounding was likely. The review therefore supports CGM as a research and phenotyping tool, not as an established treatment strategy.

Interpretation and potential mechanisms

Several mechanisms could link dynamic dysglycaemia to secondary brain injury. Glucose fluctuations may generate oxidative stress, endothelial dysfunction, inflammation and microvascular injury [26, 27]. In acute cerebral ischaemia, these processes may worsen penumbral injury, impair collateral circulation and increase blood–brain barrier permeability after reperfusion [7, 28]. Hyperglycaemia may also promote platelet activation and coagulation [29], whereas hypoglycaemia can deprive vulnerable tissue of substrate and may be difficult to recognise in patients with aphasia or reduced consciousness.

These pathways remain biologically plausible rather than directly demonstrated by the included studies. The single interventional trial did not isolate reduced variability from changes in mean glucose or other elements of care. Moreover, GV may be a marker of sympathetic activation, cortisol release, infection, fever, nutritional instability or systemic inflammation rather than an independent causal target [30]. These factors also correlate with stroke severity and outcome.

Residual confounding is therefore central to interpretation. Baseline NIHSS is a powerful determinant of outcome and can itself drive glycaemic instability through neuroendocrine stress, reduced intake and complications. Small multivariable models cannot fully separate these pathways, and statistically significant adjusted associations do not establish causality. Intervention trials must demonstrate that deliberately modifying a specified CGM metric improves patient-centred outcomes without increasing hypoglycaemia.

CGM metrics and their clinical utility

Reported metrics included mean glucose, SD, CV, mean absolute glucose, threshold-based AUC and TIR/TAR/TBR. Mean glucose is intuitive but does not describe fluctuations. CV normalises variability to the mean, while mean absolute glucose reflects rate of change. Threshold-based AUC and TAR quantify hyperglycaemic burden; TIR and TBR may be actionable but depend on the chosen limits. No metric or acute-stroke threshold has been validated as superior.

Impact of diabetes status on glycaemic variability and prognosis

Diabetes prevalence varied markedly across studies. In established diabetes, chronic hyperglycaemia, treatment exposure and autonomic dysfunction may alter both baseline glucose and variability. In patients without diabetes, stress hyperglycaemia may reflect a stronger catecholamine

and inflammatory response to stroke. The prognostic meaning of the same CGM value may therefore differ by diabetes status and HbA1c. Included studies were underpowered for interaction analyses, and the diabetes-only randomised trial cannot be generalised to stress hyperglycaemia. Future trials should prespecify diabetes status, HbA1c and glucose-lowering treatment as potential effect modifiers.

Reperfusion therapy and glycaemic variability

Three studies involved thrombectomy-treated populations [19, 20, 24]. Kersten 2024 found that admission hyperglycaemia predicted unfavourable 90-day outcome, whereas Shi 2024 and Kersten 2023 primarily assessed feasibility and accuracy. None established that CGM-guided treatment improves post-thrombectomy outcomes. Reperfusion may modify the relationship through oxidative stress, inflammatory activation and blood–brain barrier disruption, while anaesthesia, enteral feeding and critical illness can further affect glucose profiles. Future studies should stratify by intravenous thrombolysis and thrombectomy, adjust carefully for baseline NIHSS and recanalisation, and distinguish mortality from disability outcomes.

Comparison with previous literature

The findings are consistent with associations between GV and adverse outcomes in critical illness and cardiovascular disease [8, 9, 31]. Previous stroke literature has focused mainly on admission, persistent or mean glucose, while intensive glucose-lowering trials using intermittent monitoring have not shown clear functional benefit [3–6, 32]. CGM may add information by detecting excursions and hypoglycaemia that capillary testing misses, but whether this additional information changes management or outcome remains unproven.

Context within existing evidence and guidelines

AHA/ASA guidance recommends treating clearly elevated glucose to approximately 140–180 mg/dL while avoiding hypoglycaemia, and advises against intensive lowering to 80–130 mg/dL [2]. ESO guidance similarly discourages intensive insulin therapy [33]. Neither guideline incorporates CGM-derived variability metrics. The observational associations in this review indicate that patients can have clinically relevant excursions despite acceptable mean glucose, but they do not define a treatment target. Future guideline consideration would require reproducible thresholds, evidence that intervention changes outcomes and clear safeguards against hypoglycaemia.

Clinical implications

CGM should currently be considered a research tool rather than routine care. Priority populations for future trials include patients with diabetes or stress hyperglycaemia, severe stroke, and those undergoing thrombectomy, in whom glycaemic instability may be frequent and symptoms of hypoglycaemia difficult to recognise. Potential uses include phenotyping early glucose patterns, identifying high-risk patients, monitoring safety during glucose-lowering treatment and guiding development of targeted protocols. Universal CGM for all stroke admissions is not supported by current evidence.

Implementation would require validated targets, confirmatory capillary testing when readings are unexpected, safeguards against hypoglycaemia, staff training and integration with acute stroke workflows. Devices differ in sensor placement, calibration, data availability and performance during altered perfusion. Additional nursing workload, interoperability with electronic records and cost-effectiveness must also be evaluated before adoption. The absence of serious device-related events in small studies should not be interpreted as definitive evidence of safety.

Research priorities

Priorities are multicentre randomised trials of clearly defined CGM-guided protocols; validation of prognostic metrics and clinically actionable thresholds; prespecified analyses by diabetes status, HbA1c, stroke severity and reperfusion therapy; accuracy studies across the full glycaemic range; and standardised reporting of hypoglycaemia, monitoring completion, adverse events, workflow effects and cost-effectiveness. Core outcomes should include 90-day mRS, mortality, neurological deterioration and treatment-related hypoglycaemia.

Strengths and limitations

Strengths include broad database and grey-literature searching, duplicate independent screening and extraction, explicit adjudication, inclusion of feasibility and interventional evidence, validated risk-of-bias tools and outcome-level GRADE assessment. The review also distinguishes prognostic associations from treatment effects and identifies the limitations created by inconsistent CGM metrics.

Limitations include few studies, predominantly observational designs, small samples and substantial heterogeneity in populations, devices, monitoring periods, metrics and outcomes, which precluded meta-analysis. Mixed stroke cohorts and unclear CGM timing required protocol amendments. Stroke severity, physiological stress, infection,

nutrition and treatment may confound associations, and many outcomes were informed by single studies. English-language restriction and exclusion of pre-2010 studies may have introduced selection bias. Haemorrhagic transformation, cerebral oedema, long-term outcomes, workflow burden and cost-effectiveness were inadequately reported.

Conclusions

CGM appears feasible in acute ischaemic stroke and can detect glycaemic excursions missed by intermittent testing. Higher GV or hyperglycaemic exposure was associated with poorer outcomes in several observational cohorts; an unadjusted TAR difference between functional-trajectory groups did not remain independently significant after adjustment. Certainty is low or very low, and causal inference is not possible. The single randomised trial provides preliminary, short-term evidence from a multi-component intervention in patients with diabetes. Routine implementation should await standardised metrics, validated targets and adequately powered multicentre trials assessing patient-centred outcomes and hypoglycaemia.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00592-026-02766-6.

Author contributions KC conceived and supervised the review and adjudicated disagreements. JV and RPJ screened records, extracted data and completed risk-of-bias assessments. KC drafted the manuscript. All authors critically revised the manuscript and approved the final version.

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Data availability All data analysed in this review are contained within the published source articles and the accompanying supplementary material.

Declarations

Conflict of interest The authors have no relevant financial or non-financial interests to disclose.

Ethical approval and consent to participate Not applicable; this systematic review analysed data from previously published studies.

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