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Meta-Analysis

Comparative fetomaternal outcomes of acarbose versus insulin in gestational diabetes mellitus: a systematic review and meta-analysis of randomized controlled trials

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ABSTRACT

The prevalence of gestational diabetes mellitus (GDM) continues to increase globally, particularly in Southeast Asia. Currently, insulin remains the standard therapy for GDM, however its use is invasive and has been associated with hypoglycemia in up to 70% of patients. Acarbose is an alternative oral agent with minimal systemic absorption that works by inhibiting alpha-glucosidase enzyme. Therefore, this study aimed to evaluate the fetomaternal outcomes associated with acarbose therapy in pregnant women with GDM. A systematic search was conducted in PubMed, ScienceDirect, CENTRAL, Google Scholar, and MedRxiv following PRISMA guidelines. Outcomes were analyzed using mean difference (MD) and risk ratio (RR). A total of five studies involving 452 patients were included in the analysis. No statistically significant reduction in HbA1c levels was observed in either group. However, HbA1c tended to be lower in the acarbose group compared with the insulin group (MD=-0.11; 95% CI -0.24, 0.01; p=0.08). Similarly, no significant differences were found in birth weight (MD= -0.11; 95% CI -0.24, 0.01; p=0.08), macrosomia incidence (RR=1.31; 95% CI 0.62, 2.77; p=0.49), or neonatal hypoglycemia (RR=0.56; 95% CI 0.23, 1.36; p=0.20). Nevertheless, neonatal hypoglycemia occurred more frequently in the insulin group than in the acarbose group (20/138 vs 7/91, respectively). Acarbose may be a potential oral alternative for GDM management, demonstrating fetomaternal outcomes comparable to insulin. Nevertheless, future large-scale studies are required to confirm its efficacy and safety.

Keywords: Acarbose, Fetomaternal, Gestational diabetes mellitus, Insulin

INTRODUCTION

Gestational diabetes mellitus (GDM) is defined as glucose intolerance with onset or first recognition during pregnancy, particularly in the second and third trimesters.[1] The prevalence of GDM continues to increase globally and is estimated to reach 14% of the total pregnant population.[2-5] Southeast Asia has the highest prevalence of GDM (24.2%), whereas Africa has the lowest prevalence (10.5%).[6] Pregnant women with GDM have a higher risk of developing gestational hypertension and preeclampsia due to increased production of pro-inflammatory cytokines like tumor necrosis factor-alpha (TNF-α), leptin, and resistin, which lead to increased vascular resistance.[7]

In addition, hyperglycemia during pregnancy can cause fetal hyperinsulinemia and macrosomia (birth weight ≥4000 grams), with a risk occurring 2–3 times more frequently compared with normoglycemic pregnancies.[2,4] After birth, infants are at risk of neonatal hypoglycemia

due to persistent hyperinsulinemia. This condition may lead to neurological disorders such as cerebral palsy, epilepsy, and even death.[8]

Complications in patients with GDM can be reduced with good glycemic control. Insulin has become the standard therapy for GDM, particularly in cases that cannot be controlled with nutritional therapy and exercise. Insulin works by increasing glucose uptake in peripheral tissues and inhibiting glucose production and release in the liver, thereby reducing blood glucose levels.[7,9]

However, its use requires multiple daily injections and has been reported to cause hypoglycemia in 70% of patients with GDM who use insulin.[9] Meanwhile, alternative oral antihyperglycemic agents such as metformin and glyburide are currently available. However, these agents can cross the placental barrier and may potentially increase fetal complications, including neonatal hypoglycemia.[10] An alternative oral antihyperglycemic agent with lower absorption (<2%) into the maternal circulation is acarbose.[11]

Acarbose is a pseudo-carbohydrate that works by inhibiting the alpha-glucosidase enzyme. This enzyme is responsible for breaking down oligosaccharides, trisaccharides, and disaccharides into glucose and other monosaccharides, thereby delaying glucose absorption and reducing postprandial increases in blood glucose levels.[12]

Given the limitations of currently available oral therapies, acarbose may serve as an alternative oral treatment for pregnant patients with GDM. However, to date, no meta-analysis has evaluated the effectiveness and safety of acarbose in patients with GDM. Therefore, this study was conducted to compare the efficacy and safety of acarbose and insulin therapy on maternal and fetal outcomes in patients with GDM.

METHODS

Study design

This study was designed as a meta-analysis, rigorously adhering to the preferred reporting items for systematic reviews and meta-analyses (PRISMA) framework. The review protocol was prospectively registered in the International Prospective Register of Systematic Reviews (PROSPERO) with registration number CRD 420261351871, thereby strengthening the credibility of the research process.

Data sources and literature search

The systematic literature searching was conducted across five biomedical databases, including PubMed, ScienceDirect, Google Scholar, Cochrane, and MedRxiv from 30 January 2026 to 28 February 2026. The search strategy employed medical subject headings (MeSH) terms combined with Boolean operators as follows: ((“gestational diabetes mellitus” OR GDM) AND (acarbose OR “alpha-glucosidase inhibitor”) AND (insulin)).

Study selection criteria and quality assessment

To obtain eligible and high-quality literature, several inclusion criteria were applied in the selection process, including: participants: pregnant woman diagnosed with GDM; intervention: comparing acarbose oral therapy and insulin injection therapy; outcomes: reporting at least one of the following, including maternal outcome (glycemic parameters such as glycated hemoglobin/HbA1c and postprandial glucose, and mode of delivery) and/or fetal outcome (birth weight, gestational age at delivery, macrosomia, and other complications such as neonatal hypoglycaemia); study design: randomized controlled trial; language: English and Indonesian; and available in full text. While the exclusion criteria included: study with article review design, case report, or conference abstract without full text; paid full text study; and studies that did not report relevant maternal or fetal/neonatal outcomes.

The risk of bias of included studies was assessed using the revised tool for risk of bias in randomized trials (ROB2). The domains evaluated for each study, including random sequence generation, allocation concealment, insufficient outcome data, selective outcome reporting, and other potential risk of bias.

Data extraction and analysis

Data from all included studies were extracted according to the outcomes evaluated in this study, including the author’s names, year of publication, country, study design, sample size, intervention group regimen, comparator group regimen, and study outcomes. All data were systematically recorded as presented in Table 1.

The quantitative analysis in this meta-analysis was performed using Review Manager (RevMan) version 5.4.13 The result of this study was provided as means and standard deviations (SD) for the continuous variables, including HbA1c and birth weight. For dichotomous outcome, including incidence of macrosomia and neonatal hypoglycemia, risk ratios (RRs) with 95% confidence intervals (CIs) were calculated.

RESULTS

The literature search across five biomedical databases identified 12.174 studies using the predefined keywords. After removing duplicates, 10.920 studies remained and were screened based on relevant titles and abstracts. A total of 1.676 studies were further assessed for full-text availability and accessibility, resulting in 963 eligible studies. These studies were then comprehensively evaluated, yielding five RCTs that met the inclusion and exclusion criteria.[14-18]

The study selection process followed the PRISMA guidelines, as illustrated in Figure 1. All included studies reported both maternal and fetal outcomes. In total, 452 pregnant women diagnosed with GDM were included. The participants were divided into two groups which consist of intervention (acarbose) and control (insulin) group. Detailed of trials characteristic are summarized in Table 1.

Table 1: Characteristic and outcomes of included studies investigating acarbose and insulin in gestational diabetes mellitus.

Author (year)	Study design	Population (sample size)	Intervention regimen	Control regimen	Outcome
Jayasingh et al (2020)[14]	RCT	Pregnant women diagnosed with GDM (n=100)	Acarbose	Insulin	Glycemic control in patients with GDM was comparable between the acarbose and insulin groups. The mean (SD) HbA1c levels in the insulin group [6.50 (0.41)] and the acarbose group [6.42 (0.30)] showed no significant difference (p=0.82). There was also no significant difference in mean birth weight between infants born to mothers receiving either therapy (p=0.21).
Bertini et al (2005)[15]	RCT	Pregnant women with GDM who needed therapy in addition to diet and exercise programs (n=70)	Acarbose 50 mg before main meals, titrated by 50 mg weekly up to a maximum dose of 300 mg/day	Insulin therapy (rapid-acting regular insulin before meals and NPH insulin at bedtime, dose based on gestational age and body weight). Glyburide 5 mg/day, titrated up to 20 mg/day	No statistically significant differences were observed in fasting and postprandial glucose levels or in mean birth weight among the three groups. The incidence of neonatal hypoglycemia in the acarbose, insulin, and glyburide groups was 1, 1, and 8; respectively.
Villarreal-Rodriguez et al (2020)[16]	RCT	Pregnant women with GDM, who failed to achieve glycemic targets after at least two weeks of dietary intervention (n=108)	Acarbose starting at 25 mg three times daily, titrated up to 100 mg three times daily	Standard insulin therapy initiated at 0.5–0.7 U/kg/day and titrated to achieve glycemic targets	In the acarbose group, 27 of 38 subjects (71%) successfully achieved and maintained glycemic targets until delivery. No differences were observed in birth weight, gestational age at delivery, or Apgar scores. Perinatal complications were more frequent in the insulin group (5/38) compared with the acarbose group (3/38).
Sharon et al (2018)[17]	RCT	Pregnant women diagnosed with GDM between 12–32 weeks of gestation who failed to achieve glycemic control after two weeks of dietary therapy (n=83)	Acarbose 50 mg three times daily, titrated up to 200 mg three times daily	Insulin (both rapidly and intermediate acting as required by the patient)	Acarbose demonstrated comparable efficacy to insulin in controlling HbA1c levels. The mean reduction in HbA1c was 0.8454 in the acarbose group and 0.7853 in the insulin group. Fetal outcomes, including birth weight and umbilical cord blood insulin levels, were comparable

Continued.

Author (year)	Study design	Population (sample size)	Intervention regimen	Control regimen	Outcome
					between the two groups. Neonatal hypoglycemia occurred in 13 infants in the insulin group and was not observed in the acarbose group.
Veciana et al (2002)[18]	RCT	Pregnant women with GDM (n=91)	Acarbose 25 mg orally three times a day, increased to a maximum of 100 mg orally three times a day	Insulin lispro plus Neutral Protamine Hagedorn insulin (0.8 u/kg in the second trimester and 0.9 u/kg in the third trimester)	Glycemic control was reported to be similar between acarbose and insulin groups

RCT: Randomized controlled-trial, GDM: gestational diabetes mellitus, NPH: neutral protamine Hagedorn

The risk of bias for each included study was assessed using the RoB 2 tool. Based on the assessment, two study were categorized as having some concerns, while the remaining three studies were considered to have a low risk of bias. In the studies with some concerns, the final analyzed sample size was <95% of the initial sample due to loss to follow-up or participant withdrawal. Detailed risk of bias assessments for each study, as well as the summary across domains, are presented in Figure 2.

Maternal outcome

Glycemic control

Two studies were included in the analysis of HbA1c levels in patients with GDM.[14,17] The quantitative analysis reported no significant reduction in HbA1c levels in the acarbose group compared to the insulin group with the pooled effect size of MD -0.11 (95% CI -0.24 to 0.01; p=0.08) as presented in Figure 3. The forest plot showed low heterogeneity with I2=0% and p=0.35. However, in all included studies, post-intervention HbA1c levels were lower in the acarbose group compared to insulin group. In addition, the funnel plot in Figure 7A showed a symmetrical distribution across studies, indicating no apparent risk of publication bias for this outcome.

In addition to HbA1c, glycemic control was also assessed using fasting and postprandial glucose levels across the included studies. The findings consistently demonstrated comparable glycemic control between the acarbose and insulin groups. In the randomized trial by Jayasingh et al reported comparable glycemic profiles between both treatment arms throughout follow-up.[14]

Similarly, Bertini et al, no statistically significant differences were observed in fasting and postprandial glucose levels among the acarbose, insulin, and glyburide groups.[15] In the study by Villarreal-Rodriguez et al, 71% of patients in the acarbose group achieved and maintained glycemic targets without requiring insulin, further supporting its efficacy in glycemic control.[16] Consistent with these findings, Sharon et al demonstrated similar reductions in glucose parameters between groups.[17]

Meanwhile, Veciana et al also reported comparable glycemic control between the two interventions, although quantitative data were not available. Overall, these results suggest that acarbose provides glycemic control comparable to insulin when assessed using glucose-based parameters.[18]

Fetal outcome

Birth weight

Four studies were included in the analysis of neonatal birth weight among infants born to mothers with GDM.[14-17] The quantitative analysis reported no significant difference in birth weight between the acarbose and insulin groups, with the pooled effect size of MD 70.32 (95% CI -122.33 to 262.96; p=0.47) as presented in Figure 4.

High heterogeneity was observed among the included studies (I^2=62%, p=0.05); therefore, a random-effects model was applied for the analysis. In addition, the funnel plot in Figure 7B showed an asymmetric distribution across studies, indicating a potential risk of bias for this outcome.

Macrosomia

Three studies were included in the analysis of macrosomia among neonates born to mothers with GDM.[14-16] The quantitative analysis reported that the incidence of macrosomia did not differ significantly between pregnant women treated with acarbose and those treated with insulin, with the pooled effect size of RR 1.31 (95% CI 0.62 to 2.77; p=0.49) as stated in Figure 5.

Low heterogeneity was reported in this outcome (I2=0%; p=0.58). In addition, the funnel plot shown in Figure 7C demonstrated a symmetrical distribution across all studies, indicating no significant risk of publication bias.

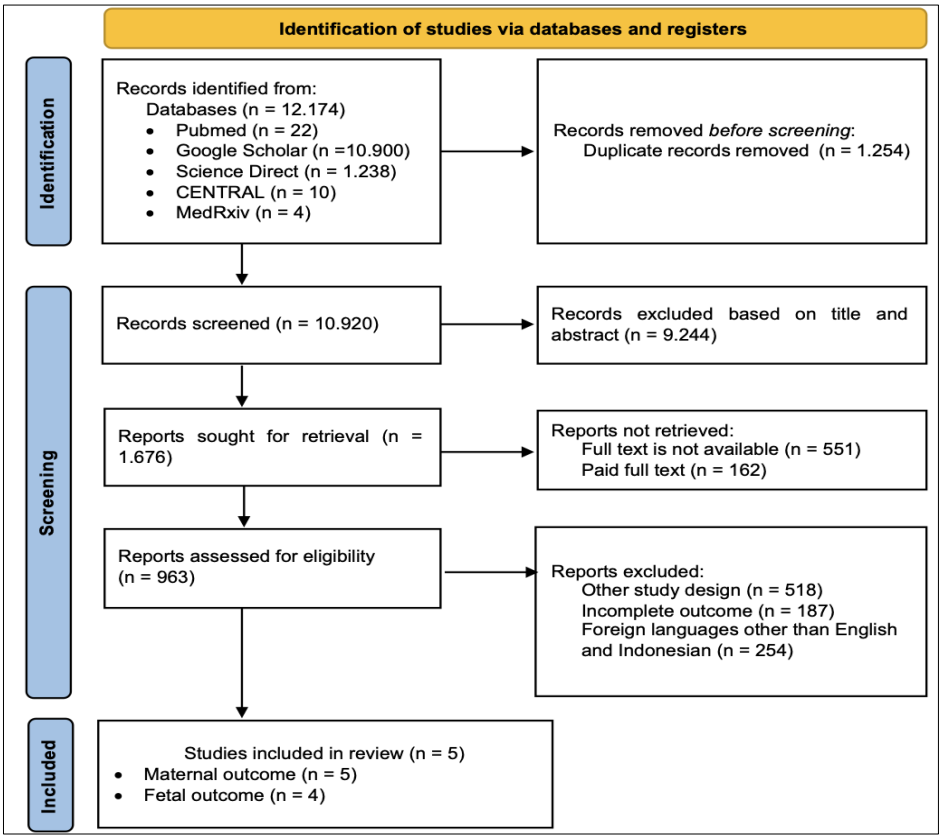

Figure 1: Flow diagram of the study selection process using PRISMA guideline.

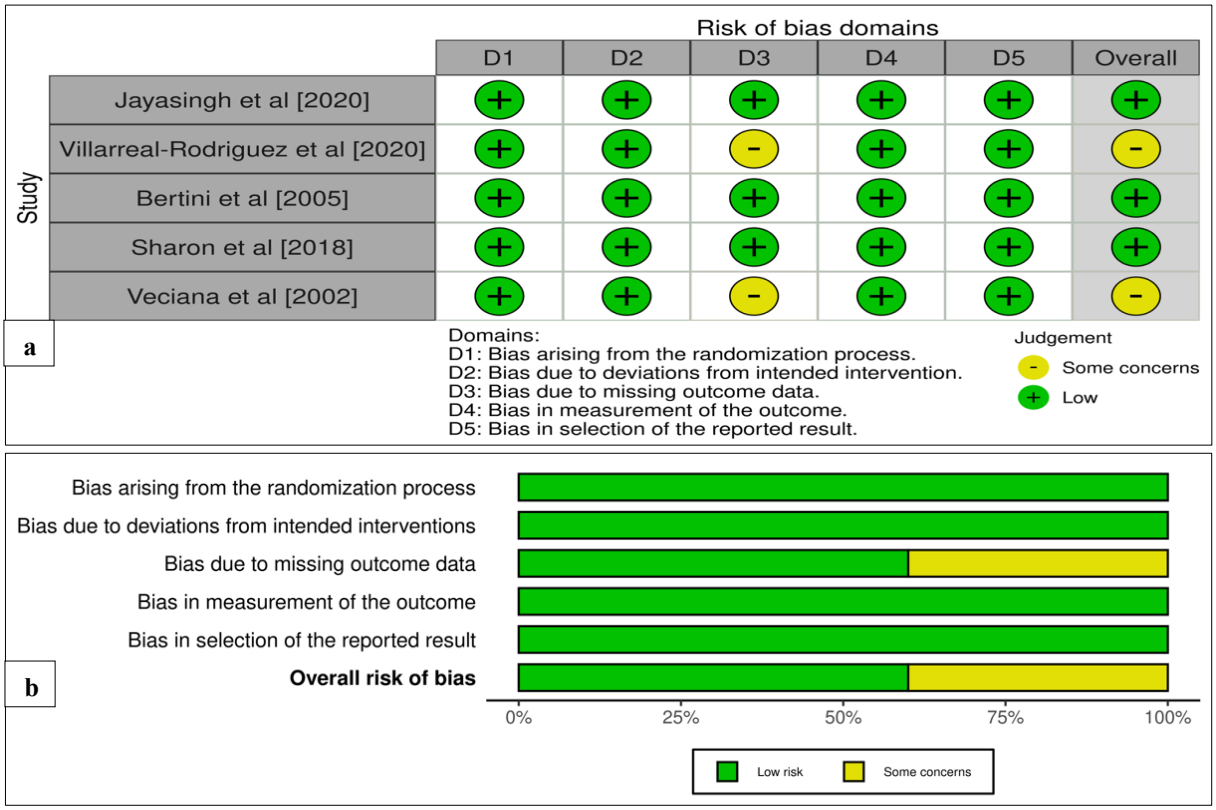

Figure 2: Risk of bias analysis of the randomize-controlled trial studies (a) traffic-light plot of the risk of bias across each domain, and (b) summarize of the risk of bias.

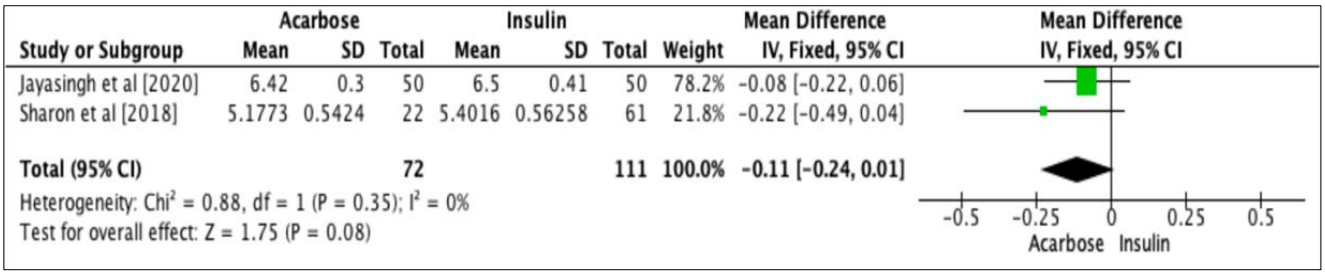

Figure 3: Forest plot of HbA1c reduction.

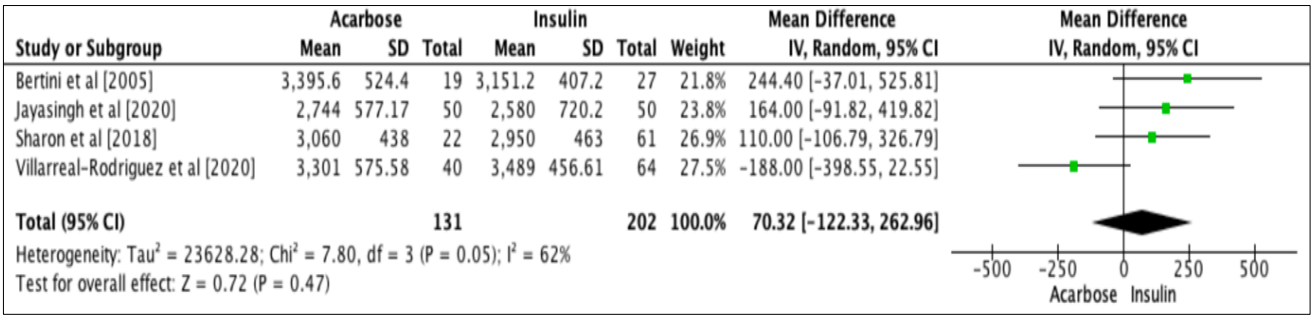

Figure 4: Forest plot of birth weight.

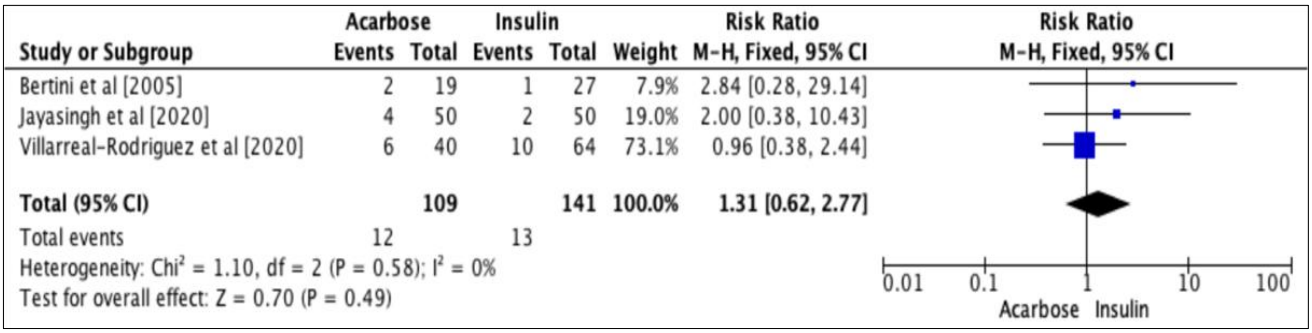

Figure 5: Forest plot of macrosomia events.

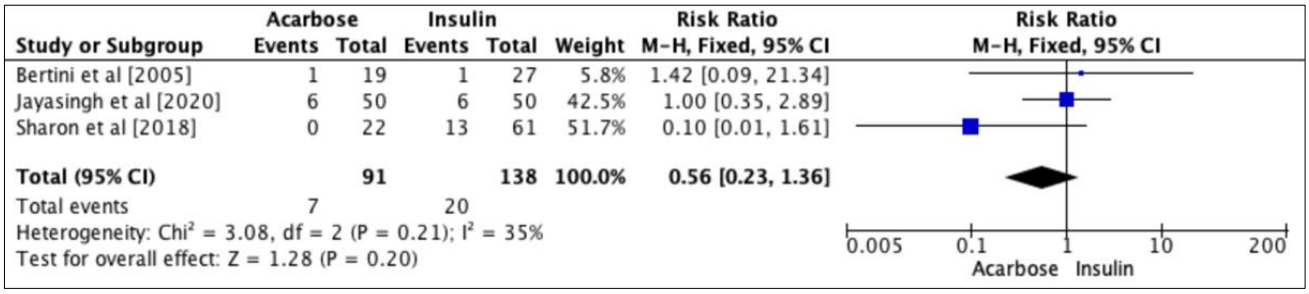

Figure 6: Forest plot of neonatal hypoglycemia.

Neonatal hypoglycemia

Three studies were included in the analysis of neonatal hypoglycemia among infants born to mothers with GDM.[14,15,17] The quantitative analysis showed that the incidence of neonatal hypoglycemia did not differ significantly between pregnant women treated with acarbose and insulin, with the pooled effect size of RR 0.56 (95% CI 0.23 to 1.36; p=0.20), as presented in Figure 6. However, neonatal hypoglycemia occurred more frequently in the insulin group than in the acarbose group (20/138 versus 7/91, respectively). Moderate heterogeneity was reported in this outcome (I2=35%, p=0.21). Furthermore, the funnel plot presented in Figure 7D demonstrated a symmetrical distribution across all studies, indicating no significant risk of publication bias.

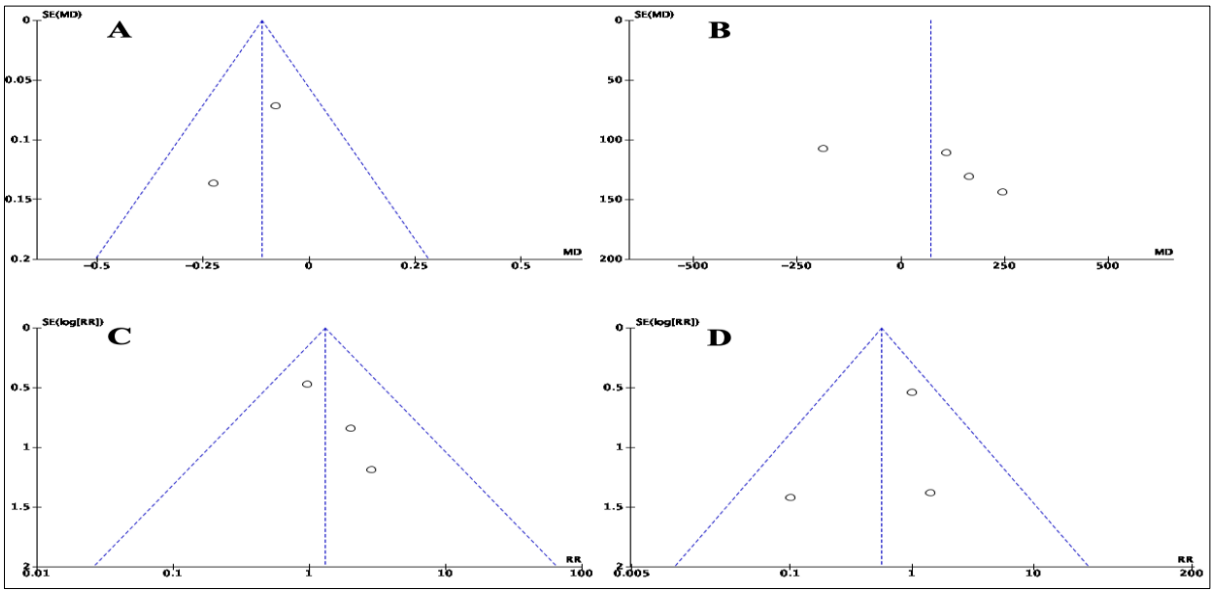

Figure 7: Funnel plots for publication bias (A) HbA1c reduction, (B) birth weight, (C) macrosomia, and (D) neonatal hypoglycemia.

DISCUSSION

This meta-analysis demonstrates that the efficacy and safety of acarbose and insulin on fetomaternal outcomes are not significantly different. Acarbose therapy showed better outcomes in reducing HbA1c levels and decreasing the incidence of neonatal hypoglycemia. In contrast, insulin therapy demonstrated better outcomes in preventing macrosomia.

Acarbose is an oral antidiabetic agent that regulates blood glucose levels by inhibiting pancreatic alpha-amylase (PAA) and intestinal alpha-glucosidase enzymes. PAA is responsible for the breakdown of complex carbohydrates into oligosaccharides, while alpha-glucosidase further hydrolyzes oligosaccharides, trisaccharides, and disaccharides into monosaccharides such as glucose and fructose. Inhibition of these enzymes delays carbohydrate digestion and absorption, thereby attenuating postprandial glucose excursions.[19,20] The delayed digestion of carbohydrates also enhances the secretion of glucagon-like peptide-1 (GLP-1), which contributes to slower gastric emptying, modulation of insulin secretion, and suppression of glucagon release. These effects collectively support improved fasting glycemic control.[21,22] In addition, acarbose has been shown to reduce pancreatic beta-cell stress and hyperinsulinemia. As blood glucose levels decrease, insulin secretion is correspondingly reduced, thereby mitigating hyperinsulinemia commonly observed in individuals with prediabetes and early-stage diabetes.[20,22]

Maternal outcome

Based on the analysis in this study, HbA1c levels did not differ significantly between patients treated with acarbose and those receiving insulin therapy. However, across the included studies, the mean post-intervention HbA1c levels tended to be lower in the acarbose group compared with the insulin group. Acarbose monotherapy has been reported to reduce HbA1c by approximately 0.5% to 0.8%. In a retrospective study, a significant reduction in HbA1c of 0.49% (p=0.012) was observed, with mean HbA1c decreasing from 8.97% (SD=1.75) before treatment to 8.48% (SD=1.82) after acarbose therapy.[23] Although no statistically significant difference in HbA1c levels was observed between the acarbose and insulin groups, the consistently lower post-treatment HbA1c values reported in the included studies suggest that acarbose provides glycemic control comparable to insulin therapy.

All studies included in this meta-analysis administered acarbose using varying dosing regimens. A placebo-controlled trial evaluated the reduction in HbA1c using three different daily doses of acarbose (100 mg, 200 mg, and 300 mg) compared with placebo in patients with type 2 diabetes mellitus. The study demonstrated significant mean reductions in HbA1c of 0.78%, 0.73%, and 1.10% in the 100 mg, 200 mg, and 300 mg three-times-daily groups, respectively.[24] Although the 300 mg regimen achieved the greatest reduction, the maximum daily dose approved by the Food and Drug Administration (FDA) is 100 mg administered three times daily.[22]

In addition to HbA1c, postprandial glucose plays a crucial role in overall glycemic control, particularly in patients with GDM. Across the included studies, no significant differences in postprandial glucose levels were observed between the acarbose and insulin groups, indicating comparable efficacy in controlling postprandial glycemia. This finding is biologically plausible, as acarbose exerts its primary effect by inhibiting α-glucosidase enzymes in the intestinal brush border, thereby delaying carbohydrate digestion and absorption, leading to a reduction in

postprandial glucose excursions. Evidence from patients with type 2 diabetes mellitus further supports this mechanism, where acarbose has been shown to significantly reduce postprandial glucose levels and glycemic variability. Previous studies have demonstrated that acarbose can lower postprandial glucose excursions by approximately 30–50 mg/dl, depending on the dose and baseline glycemic status.[25,26]

Fetal outcome

Maternal hyperglycemia leads to increased transplacental glucose transfer to the fetus, resulting in pancreatic β-cell hyperplasia and fetal hyperinsulinemia. After delivery, persistent fetal hyperinsulinemia, combined with the abrupt cessation of maternal glucose supply, leads to neonatal hypoglycemia. In mothers with GDM, the risk of neonatal hypoglycemia increases by three- to twelve-fold. Clinical manifestations of neonatal hypoglycemia include tremors, lethargy, seizures, and respiratory distress.[27,28]

The incidence of neonatal hypoglycemia in all included studies did not differ significantly between the acarbose and insulin groups; however, hypoglycemia occurred more frequently in the insulin group than in the acarbose group (14/88 versus 1/41, respectively). In one retrospective study, insulin use was reported as the only predictive factor for the occurrence of neonatal hypoglycemia.[29]

Poor maternal glycemic control during pregnancy is one of the risk factors for neonatal hypoglycemia. Multiple logistic regression analysis in a study by Cao et al demonstrated a significant association between maternal glycemic control and neonatal hypoglycemia ($p=0.001$). The mean postnatal blood glucose levels in neonates born to mothers with HbA1c ≥6% before delivery were lower compared to those with HbA1c<6% ($p<0.05$). This results in disturbances in neonatal glucose metabolism that are significantly associated with the occurrence of neonatal hypoglycemia. It can be concluded that higher maternal HbA1c levels increase the risk of neonatal hypoglycemia.[30]

In this study, no significant differences were found in birth weight and the incidence of macrosomia between the insulin and acarbose groups. All included studies consistently showed no significant differences.[14-17] The incidence of macrosomia has been reported to increase two- to five-fold in pregnant women with GDM, particularly in those with poor postprandial glycemic control.[31] Furthermore, Cao et al also reported that pre-delivery HbA1c levels significantly influenced birth weight ($p=0.005$).[30]

Macrosomia is caused by fetal hyperglycemia. Maternal glucose crosses the placenta, whereas maternal insulin does not. Consequently, fetal hyperglycemia stimulates pancreatic β-cell hyperplasia and results in fetal hyperinsulinemia. This condition promotes accelerated fetal growth and enlargement of several organs, including the liver, spleen, and heart, thereby leading to macrosomia. Therefore, infants born to mothers with GDM may have normal birth weight when maternal glycemic control is achieved.[32]

Overall, the findings of the present study, together with previous evidence, suggest that acarbose provides fetomaternal outcomes comparable to insulin therapy. The oral route of administration may further enhance treatment acceptability, making acarbose a promising alternative for GDM treatment.

Limitations

Several limitations of this study should be considered when interpreting the findings. First, the number of included randomized controlled trials was limited, resulting in relatively small sample sizes. Second, differences in acarbose dosing regimens and participant characteristics among the included studies could have introduced clinical heterogeneity. Therefore, further large-scale, multicenter randomized controlled trials are needed to provide more robust evidence regarding the efficacy and safety of acarbose in the management of GDM.

CONCLUSION

This meta-analysis indicates that acarbose and insulin have comparable efficacy and safety in the management of GDM, with no significant differences in overall fetomaternal outcomes. Acarbose demonstrated a potential effect to achieve better glycemic control and a lower incidence of neonatal hypoglycemia. These findings suggest that acarbose may serve as a potential alternative to insulin in selected patients, particularly those with predominant postprandial hyperglycemia. However, further large-scale randomized controlled trials are required to confirm these findings and to evaluate long-term maternal and neonatal outcomes.

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