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Original Research Article

Association of empagliflozin treatment with changes in the metabolic score for insulin resistance in Iraqi adults with type 2 diabetes: a 6-month prospective cohort study

Rawnaq Adil Aladhab*

Department of Pharmacology and Toxicology, College of Pharmacy, University of Basrah, Basrah, Iraq

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***Correspondence:**
Dr. Rawnaq Adil Aladhab,
Email: rawnak.yassin@uobasrah.edu.iq

ABSTRACT

Background: The metabolic score for insulin resistance (METS-IR) is an insulin-free surrogate that combines fasting plasma glucose, triglycerides, body mass index, and high-density lipoprotein cholesterol. Evidence on its responsiveness to sodium-glucose cotransporter 2 (SGLT2) inhibition is limited.
Methods: In this 6-month prospective observational cohort study, 124 adults with type 2 diabetes were enrolled at Al-Yarmouk Teaching Hospital, Baghdad. Empagliflozin 10 mg/day was added to usual oral therapy in 62 patients according to routine prescribing and patient preference; 62 continued therapies without an SGLT2 inhibitor or glucagon-like peptide-1 receptor agonist. Complete-case analyses included 58 and 56 participants, respectively. The primary outcome was the between-group difference in change in METS-IR.
Results: At 6 months, mean METS-IR decreased from 48.6±6.2 to 42.8±5.8 in the empagliflozin group and from 49.1±6.5 to 48.5±6.4 in controls. The reported adjusted between-group difference was −5.42 points (95% CI −6.81 to −4.03; $p<0.001$). Empagliflozin exposure was also associated with larger reductions in HbA1c, body weight, body mass index, and triglycerides and a larger increase in HDL cholesterol. Within the empagliflozin group, change in METS-IR correlated with changes in HbA1c ($r=0.48$), triglycerides ($r=0.41$), and body mass index ($r=0.35$), but not fasting plasma glucose ($r=0.18$).
Conclusions: Empagliflozin initiation was associated with a clinically and statistically larger reduction in METS-IR over 6 months than continued non-SGLT2 therapy. Because METS-IR contains several treatment-responsive components and treatment allocation was nonrandom, the findings should be interpreted as evidence of metabolic improvement rather than direct proof of enhanced insulin sensitivity.

Keywords: Empagliflozin, METS-IR, Insulin resistance, Type 2 diabetes, SGLT2 inhibitor, Iraq, Prospective cohort

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is defined by progressive β-cell failure over time with insulin resistance. The hyperinsulinemic–euglycemic clamp is a reference method for the direct quantification of insulin action but is too complex for routine use.[1] An insulin-dependent measure of insulin sensitivity, HOMA-IR along with other fasting indices are less but nevertheless useful.[2,3]

METS-IR was created to be an insulin-free surrogate using routinely available measurement: fasting plasma glucose (FPG), triglycerides (TG), body mass index (BMI), and high-density lipoprotein cholesterol (HDL-C) According to a derivation study, it is associated with insulin sensitivity, visceral adiposity and incident T2DM.[4] As further cohorts demonstrated, a higher METS-IR was associated with cardiovascular events, chronic kidney disease, and all-cause or cardiovascular mortality.[5-7] While these associations support prognostic significance, they do not confirm that treatment-related within-person variations

in METS-IR exactly reproduce clamp-measured changes in insulin sensitivity.

Empagliflozin reduces levels of plasma glucose through interference with renal SGLT2 stimulating increased urinary glucose excretion. Cardiovascular and kidney advantages have been confirmed through extensive outcome trials among T2DM, heart failure and chronic kidney disease populations.[11,12,16-18] Smaller mechanistic studies suggest that glucotoxicity and β-cell function may improve; effects on insulin sensitivity appear less robust.[8,9]

Empagliflozin also modifies multiple components of METS-IR. Studies irrefutably demonstrate that there is weight loss and body compositional changes with SGLT-2 inhibitors. However, there are variations in triglycerides, HDL-C, and LDL-C values with population and study design.[21-23,26,27] Thus, METS-IR may be a potentially clinically useful integrated response marker, but its mathematical coupling to treatment-responsive components must be interpreted with caution.

Although T2DM, obesity, and atherogenic dyslipidemia frequently coexist in certain populations, evidence from clinical populations in the Middle East is limited. We therefore assessed whether the initiation of empagliflozin was associated with a greater 6-month change in METS-IR than continuation of oral glucose-lowering therapy without an SGLT2-inhibitor. We looked into anthropometric, glycemic, and lipid changes in the same period along with change in METS-IR androidx its metabolic correlates.

METHODS

Study design and setting

A prospective, two-group observational cohort study was conducted in the Endocrine and Diabetology Center of Al-Yarmouk Teaching Hospital, Baghdad, Iraq, from January 2023 to January 2024. Follow-up was 6 months. Reporting was structured according to STROBE principles for observational study.[28] The Institutional Review Board of the College of Medicine, University of Baghdad, was reported to approve the protocol (approval no. 142/2022) and reported written informed consent for all participants. The letter of approval must be compared with the originals for submission.

Participants

Eligible participants included adults aged 30-65 years with 1 year of diagnosis of T2DM, HbA1c of 7.5-10.0% despite metformin and/or sulfonylurea therapy, and willingness to adhere to the recommendations of the study investigators. Participants who were excluded from the study were those with type 1 diabetes; estimated glomerular filtration rate less than 45 ml/min/1.73 m²; recurrent urinary tract infection; severe hepatic impairment; history of diabetic ketoacidosis; pregnancy; and lactation.

Exposure definition and comparator

Allocation of treatment was not randomised. The exposed group consisted of patients who were prescribed empagliflozin 10 mg daily in addition to their baseline oral therapy. The comparator group received oral therapy without an SGLT2 inhibitor or GLP-1 receptor agonist. Distribution was reflective of standard clinician prescribing, availability, cost and patient preference. The treating physician could change oral medicine used in conjunction with IV iron. The design estimates an association with starting treatment in real-world conditions and is subject to confounding by indication and access.

Clinical and biochemical measurements

At baseline and six months, the anthropometry was measured with the participant dressed in light clothing and not wearing shoes; BMI was calculated as kg/m². Blood pressure was measured as a part of routine clinical assessment. Following a 10–12-hour overnight fast, the measurement of FPG, total cholesterol, TG, and HDL-C was done on Cobas c 501 analyser (Roche Diagnostics, Germany) using enzymatic colourimetric assays. A high-performance liquid chromatography was used to measure HbA1c (Bio-Rad D-100). When TG was <400 mg/dl, LDL-C was calculated using the Friedewald equation, as per the measurement approach outlined in the reference dataset.[29]

Outcome definition

The pre-specified primary outcome for this manuscript was the between-group difference in change in METS-IR from baseline to 6 months. METS-IR was calculated as given.

$$METS - IR = ln[(2 \times FPG) + TG] \times BMI / ln(HDL - C)$$

FPG, TG, and HDL-C were expressed in mg/dl and BMI in kg/m². Secondary outcomes were changes in HbA1c, body weight, BMI, TG, and HDL-C. The supplied aggregate dataset did not contain analyzable adverse-event data; safety was therefore not evaluated.

Statistical analysis

To examine the distribution of continuous variables, we performed the Shapiro–Wilk test. The other variables were summarized as mean±SD or median (interquartile range) based on their distribution. Baseline comparisons between groups were done using the independent-samples t-test or Mann–Whitney U test, as appropriate. Categorical variables used χ^2 tests. Longitudinal comparisons were reported as repeated-measures analysis, or ANCOVA adjusted for baseline values and concomitant sulfonylurea use. Correlations were assessed using either Pearson or Spearman coefficients, whichever appropriate. For our

analyses, we used two-sided alpha, which amounted to 0.05.

The complete-case analysis of participants with a follow-up at 6 months was possible for this reconstruction. The absence of participant-level data hindered any capability to reproduce any sort of multiple imputation, propensity-score analysis, inverse-probability weighting, sensitivity analysis for loss to follow-up, or correction for multiplicity. Accordingly, the adjusted primary estimate is reported as exactly provided, and all figures in this manuscript rely only on the aggregate values in the source file.

RESULTS

Participant flow

We randomly assigned 124 participants (62 per group) to the trial. Four patients assigned to empagliflozin and six to placebo did not contribute 6-month outcome data. This resulted in a complete-case population of 114 (91.9% retention) 58 empagliflozin exposed and 56 controls.

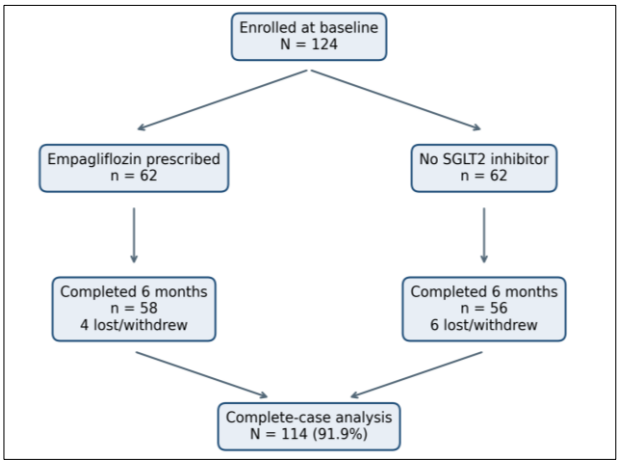

Figure 1: Participant flow and complete-case analysis population.

Baseline characteristics

The groups had similar baseline characteristics as reported. The mean age of participants was about 52 years, approximately half were male, mean BMI was about 30 kg/m², and mean HbA1c was 8.4–8.5%. None of the reported baseline comparisons reached statistical significance (Table 1).

Glycemic and anthropometric outcomes

Empagliflozin treatment (as compared with placebo) was also associated with alterations that favourably impacted HbA1c, body weight and BMI at 6 months. The reported p values were less than zero-point zero zero one for each outcome as seen in Table 2. The aggregate table provided has no follow-up FPG values and is therefore not reported as an efficacy endpoint.

Lipid profile and METS-IR

The empagliflozin group experienced a substantially lower median TG reduction than the control group. The average increase of HDL-C was 2.7 mg/dl. Mean METS-IR decreased by 5.8 versus 0.6 points. The average difference in mean change calculated without any adjustments stood at −5.2 points, whereas the other provided analysis gave an average difference in mean change of −5.42 points (95% CI −6.81 to −4.03; p<0.001).

Correlations with change in METS-IR

The changes in METS-IR correlated positively with HbA1c (r=0.48, p=0.001), TG (r=0.41, p=0.001) and BMI (r=0.35, p=0.007) in the empagliflozin group. The relationship with the FPG change was not statistically significant (r=0.18, p=0.174). Due to the presence of parameters, including BMI, TG, HDL-C, and FPG, in the METS-IR equation, the associations involving those parameters are partly dependent on mathematical "coupling" and should not be interpreted as independent mechanistic validation.

Table 1: Baseline demographic and clinical characteristics of the complete-case population.

Characteristic	Empagliflozin (n=58)	Control (n=56)	P value
Age, years	52.1±8.4	52.8±7.9	0.652
Male sex, N (%)	31 (53.4)	28 (50.0)	0.714
T2DM duration, years	6.5±3.2	7.1±3.8	0.341
BMI, kg/m²	30.2±4.1	30.5±4.3	0.708
Systolic BP, mmHg	132.4±14.2	134.1±15.0	0.523
HbA1c, %	8.4±0.6	8.5±0.7	0.412
FPG, mg/dl	158.2±32.4	161.5±35.1	0.594
Triglycerides, mg/dl	182 (145–215)	187 (150–220)	0.681
HDL-C, mg/dl	38.5±6.2	37.9±5.8	0.589
LDL-C, mg/dl	112.4±24.5	114.2±26.1	0.702
METS-IR	48.6±6.2	49.1±6.5	0.674

Data are mean±SD, median (IQR), or N (%). BP: blood pressure; FPG: fasting plasma glucose; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; T2DM: type 2 diabetes mellitus

Table 2: Six-month changes in glycemic and anthropometric outcomes.

Outcome	E: base	E: 6 mo	E: Δ	C: base	C: 6 mo	C: Δ	Between p
HbA1c, %	8.4±0.6	7.3±0.5	−1.1±0.3	8.5±0.7	8.1±0.6	−0.4±0.4	<0.001
Weight, kg	84.5±11.2	81.2±10.8	−3.3±1.5	85.1±12.0	85.8±12.4	+0.7±1.8	<0.001
BMI, kg/m²	30.2±4.1	29.0±3.9	−1.2±0.5	30.5±4.3	30.7±4.4	+0.2±0.6	<0.001

Values are mean±SD. E: empagliflozin; C: control; Δ: observed within-group change. The p value is the reported between-group comparison

Table 3: Six-month changes in lipid profile and METS-IR.

Outcome	E: base	E: 6 mo	E: Δ	C: base	C: 6 mo	C: Δ	Between p
Triglycerides, mg/dl	182 (145–215)	148 (120–175)	−34 (−45 to −22)	187 (150–220)	180 (145–210)	−7 (−15 to +2)	<0.001
HDL-C, mg/dl	38.5±6.2	41.2±5.9	+2.7±1.8	37.9±5.8	38.1±6.0	+0.2±1.5	<0.001
METS-IR	48.6±6.2	42.8±5.8	−5.8±2.1	49.1±6.5	48.5±6.4	−0.6±1.9	<0.001

Triglycerides are median (IQR); other outcomes are mean±SD. E: empagliflozin; C: control; Δ: observed within-group change

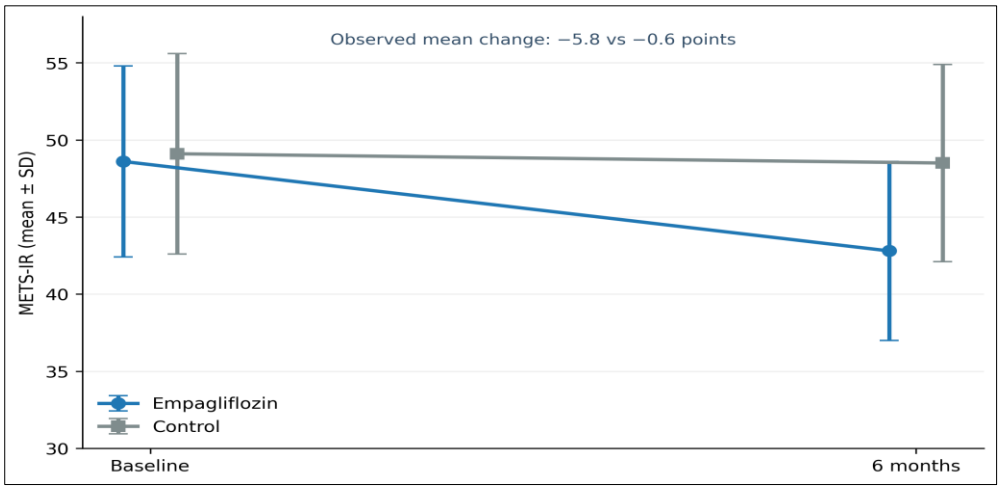

Figure 2: Mean METS-IR at baseline and 6 months by treatment group.
Error bars show SD, not confidence intervals. The figure is reconstructed from the reported aggregate values and does not represent paired participant-level trajectories

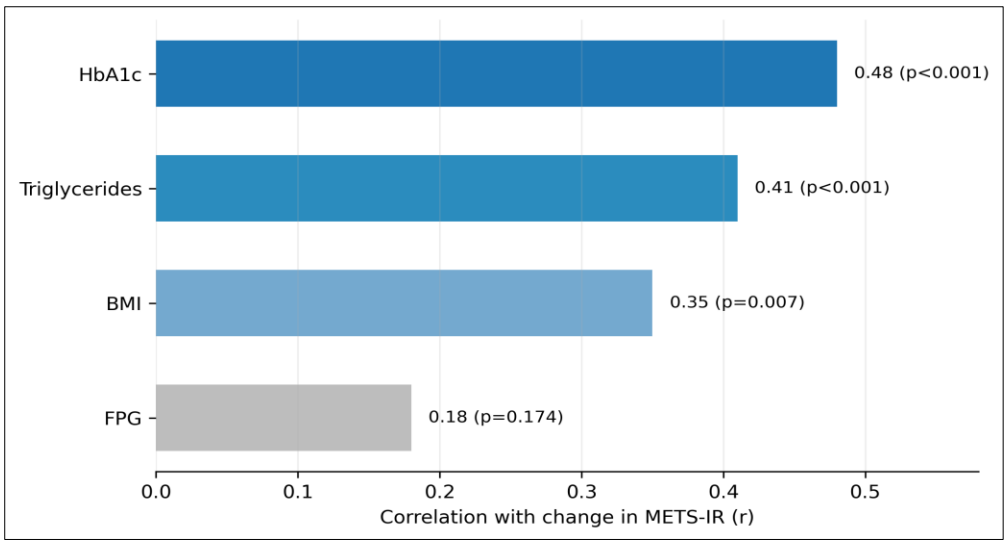

Figure 3: Reported correlations between change in METS-IR and metabolic changes in the empagliflozin group.
Positive r values indicate that larger reductions in METS-IR co-occurred with larger reductions in the listed variable as coded in the source analysis. Values are reproduced from the supplied aggregate results

DISCUSSION

Principal findings

In a future cohort of Iraq, the initiation of empagliflozin was associated with a significantly greater 6-month reduction in METS-IR compared to oral therapy without SGLT2 inhibitor. There was consistent change direction of HbA1c, body weight, BMI, TG, and HDL-C. The research findings indicate that METS-IR responds to combined glycemic, anthropometric and lipid changes observed with SGLT2 inhibition in routine practice.

A responsive metabolic score and direct measurement of insulin-sensitivity are two different things. The authors were forced to use the METS-IR surrogate to estimate insulin sensitivity. Empagliflozin alters glucose equilibrium and frequently reduces body mass; both effects enter the score.

As such, a decrease in METS-IR post-therapy cannot solely prove drug-induced enhancement of clamp-determined insulin action.

Comparison with previous evidence

The changes in HbA1c and body-weight seen in this instance are consistent with randomized evidence of glycemic and weight benefits of empagliflozin when used alone or in combination with other glucose-lowering drugs.[19,20] According to a meta-analysis of randomized trials, SGLT2 inhibitors help in reducing total body mass (TBM) and fat mass (FM).[26] It is biologically plausible that these changes are linked to METS-IR.

The lipid findings need more nuance. According to the meta-analysis, there were small class-level increase in HDL-C as well as LDL-C with TG reductions but there is considerable heterogeneity present.[27] Studies of empagliflozin have shown a reduction in triglycerides in selected phenotypes as well as changes in sterol handling.[21-23]

Other cohorts showed little overall change in lipids. The current group of participants began with high baseline TG and low HDL-C which may have made the METS-IR formula more responsive to lipid change.

Studies of the underlying mechanism show that relieving glucotoxicity with empagliflozin can improve β-cell glucose sensitivity while this is not uniformly demonstrated with direct clamp-based evidence for improved insulin sensitivity.[8,9]

Preclinical research has suggested a shift from carbohydrate to lipid use. However, it is not possible to assume a translation of that mechanism to a numerical METS-IR change. Present observations are findings complement mechanistic measurement but not replacing them.

Clinical and research implications

The use of METS-IR is recommended when there are no available measurements of fasting insulin. METS-IR is also useful for longitudinal risk profiling in regions with resource constraints. The test has calculated quantity is already obtained from tests used for diabetes care. It has no additional laboratory cost. Furthermore, it may summarize multidomain metabolic response in a single value. The strongest function of this monitoring tool may be serving as a practical monitoring or stratification device and not as a diagnostic replacement for clamp-derived insulin sensitivity.

As the outcome of interest, future trials for METS-IR must be pre-registered either as primary or secondary outcome; a randomized design or careful emulation of target-trial design must be adopted; paired measurements must be retained at the participant level; and changes in METS-IR must be compared with those of HOMA-IR, triglyceride-glucose index or direct measures of insulin sensitivity. The analyses that are predetermined must check for differences based on sex, obesity at the beginning, TG/HDL, and kidney function. It is also important to report safety, adherence, drug switching, and reasons for loss to follow-up.

Strengths and limitations

Justification of a good study design including the prospective follow-up, a clinically relevant Iraqi population, use of routinely available biochemical measures, an active comparator and an internally consistent change across the multiple metabolic domain. The higher the follow-up proportion, the less worry about attrition.

One cannot make inference because of several limitations. A patient's preference for first-line treatment is likely to affect it more than randomization. Socioeconomic status, adherence, baseline risk, and health-seeking behavior may correlate with cost and availability. In addition, the source dataset did not provide participant-level values, propensity adjustment, thorough background medication distributions, or any details about statin use, renal outcomes, adherence or adverse events. If a dataset is missing values of the outcome or exposure, then complete-case analysis can be biased. Fourth, they did not measure clamp and fasting insulin, so did not directly establish change in true insulin sensitivity. Correlations noted between changes in METS-IR and variables included in the formula were due to mathematical coupling. The generalizability and long-term interpretation of the findings is limited by a single tertiary center and six-month follow-up. The rationale for sample size and replication were unavailable.

CONCLUSION

In adults with T2DM treated at a tertiary center in Baghdad, initiation of empagliflozin was associated with greater 6-

month reduction in METS-IR than continued oral non-SGLT2 therapy. These results were accompanied by beneficial changes in HbA1c, body weight, BMI, TG and HDL-C. According to research METS-IR is a feasible integrated metabolic-response marker in settings where insulin assays are available. However, the observational design and composite nature of the score prevent causal claims and direct evidence of improved insulin sensitivity. Multicenter studies that randomize or rigorously adjust are necessary with participant-level validation.

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